THE NORMAL AND PATHOLOGICAL
HISTOLOGY OF THE MOUTH

VOLUME II
PATHOLOGICAL HISTOLOGY

The Normal and Pathological Histology of the Mouth

BEING THE SECOND EDITION OF

The Histology and Patho-Histology

OF THE

Teeth and Associated Parts

REVISED AND ENLARGED

BY

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VOLUME II

PATHOLOGICAL HISTOLOGY

WITH 394 ILLUSTRATIONS
IN THE TEXT, INCLUDING 343 ORIGINAL PHOTOGRAPHS
AND PHOTOMICROGRAPHS BY THE AUTHOR

PHILADELPHIA
P. BLAKISTON'S SON & CO.
1012 WALNUT STREET

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PART I
THE DENTAL TISSUES

INTRODUCTION

In considering the many pathological conditions of the hard and soft tissues found in the oral cavity, it is convenient to arrange, group, and describe them according to an anatomical, pathological, or clinical point of view. Each method of treatment is of value, but is dependent upon the character of the work for which it is made. Thus the clinical viewpoint is of vast importance in manuals dealing with the practice of Oral and Dental Surgery, and it is obvious that the anatomical should be of interest in a book dealing with Histology and Pathology.

Hence it follows that, as far as is consistent and convenient, the arrangement of the Morbid Affections of the mouth and containing organs in this and succeeding Chapters is built upon the morphology and minute anatomy of the tissues.

Regarding the diseases of the hard dental tissues many of which are quite unique, it is necessary to point out the fact that few are confined exclusively to one tissue, but that often the surrounding, or sublying structures share them also. The whole of the arbitrary classifications hereafter adopted, in order to save confusion, must therefore be considered in this light.

In the three following and other Chapters the various lesions are placed in alphabetical order.

The subjects of dental caries and "pyorrhæa alveolaris" have been specially relegated to Chapters by themselves.

As far as the diseases of the soft Oral and Dental tissues are concerned, they differ essentially in no characteristics from morbid conditions of other cellular and fibrous structures. The phenomena of inflammation proceed on identical lines with those elsewhere. The structure of tumours of the jaws is in agreement with that of those which affect other regions of the body. In consequence, however, of their environment, the usual histological and pathological appearances are modified to greater or less degrees, and hence are peculiarly interesting and important.

THE PATHOLOGICAL CONDITIONS OF THE ENAMEL

MICROSCOPICAL ELEMENTS IN:—(i) Hypoplasia; (ii) Enamel nodules; (iii) Pathological pigmentation; (iv) Nanoid enamel; (v) Rachitic enamel; (vi) Syphilitic enamel; (vii) Abrasion; (viii) Absorption; (ix) Attrition; (x) Erosion; (xi) Fungoid excavation.

A.—DEVELOPMENTAL DISEASES

(i) *Hypoplasia*

Definition.—Enamel, the surface of which is more or less covered with pits, or fissures, not due to absorption. The term indicates fairly accurately the condition in which the enamel is not only reduced in amount and thickness, but is developmentally defective. It is the antithesis of hyperplasia, where, as in the case of cementum, the tissue is increased in bulk. The old term, "honeycombed teeth," is falling into desuetude, as it should do, inasmuch as it is an incorrect and misleading expression. Hypoplasia was originally called by Salter[1] "Rocky" enamel. The condition may be (i) general or (ii) local.

Etiology.—The factors concerned in the production of (A) General hypoplasia of the enamel are not absolutely determined. Most probably (i) the exanthematous fevers, when sufficiently acute, particularly measles and scarlet fever, occurring during the first and second year after birth, are the chief. The pathological changes have also been ascribed (ii) to the effect produced on the ameloblasts by the exhibition of mercurial salts for the relief or cure of convulsions during the dentition of the child. (iii) Lamellar cataract is often accompanied by hypoplasic enamel. The condition is more likely to be occasioned (iv) by malnutrition from improper dietary. At all events, it is certain that it is due to modified function or altered metabolism on the part of the ameloblasts when they are most actively engaged in depositing lime salts, probably by the process of secretion. This would account for the granular

[1] Salter's "Dental Pathology and Surgery," p. 74, 1874.

appearance of the rods to be presently noticed. Exactly how the indentations or crevices themselves are formed is not known: it may be that some perverted state of Nasmyth's membrane may cause them.

Suppuration around the root or roots of deciduous teeth may induce, (B) Local manifestations of the disease in the crowns of their permanent successors.

Hypoplasia of the enamel is said to occur in deciduous teeth,[1] but care should be taken to discriminate between hypoplasia and absorption of this tissue.

FIG. 1.—Vertical section of a cusp of a molar with hypoplasia of the enamel. Prepared by grinding. Unstained. Magnified 45 times. E. Enamel; F. Fissure; P. Pit extending to the margin of the dentine; D. Dentine.

Macroscopical Appearances.—The most prominent features of hypoplasia of the enamel are loss of substance, deepening of the normal pits and fissures of its surface in situations such as the pre-molars and molars where they exist normally, pigmentation, and loss of lustre. The normal amount of the enamel is considerably

[1] Otto Zsigmondy, "Beitrage zur Kenntniss der Entstehungsursache der Hypoplastischen Emaildefecte." *Trans. World's Columbian Dental Congress*, p. 48, 1894.

FIG. 2.—Sagittal section of an incisor. Prepared by grinding. Unstained. Magnified 45 times. E. Deeply pigmented hypoplasic enamel; I.L. "Interruption lines;" F₁. Fissure or pit on labial surface; F₂. Pit on lingual surface; D. Dentine. *Cf*. Fig. 12, Vol. I.

reduced in extent. In the molars, as well as in other teeth, a pronounced line of demarcation often divides the ill from the well-developed. The shrunken appearance of the crowns of molars seems to heighten the effect of the elevation of the cusps, several of which may be raised to pointed conical eminences. The enamel, in addition to loss of its characteristic appearance[1] may be pigmented a brown or yellow colour.

In the incisors and canines the pits or grooves are sometimes very numerous and small, running either in a vertical or a coronal direction. Sometimes they are distributed uniformly over the crown, and give it a spotted aspect.

The extent of the hypoplasia varies. The commonest condition is that the crown of the first permanent molars, incisive edges and part of the crowns of the incisors, and the cusps of the canines are affected, the degree in each case of severity being similar, but of extent unequal.[2]

Secondary Changes.—There may be no secondary changes, the enamel undergoing no further alterations. But usually, marked pigmentation occurs, and caries attacking the depressions, especially when the dentine is exposed at the base of the cavities, may lead to fracture and loss of the tissue. "Arrested caries" may also occur.

HISTOLOGY

The most striking appearance, as viewed under low power, is the deeply pigmented condition of the enamel. Dense bands of brownish colour extend throughout its substance, being particularly pronounced in the neighbourhood of the breaches of surface (Fig. 1). Brown and grey stripes—"interruption lines" alternate, with little or no attempt at regularity (Fig. 2). The brown striæ of Retzius, in the majority of sections, are wanting, as also are Schreger's lines.

A diminution in the normal width of the enamel is a remarkable

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[1] As a matter of fact, the enamel is often quite white, shiny, and smooth in the areas between the depressions.

[2] In this connection it is of interest to recall the facts that "calcification centres" appear in the tooth germs of the first permanent molars about the eighth month of intra-uterine life, in the incisors about the first year of post natal life, and in the canines about the third year of post natal life. For detailed accounts of the calcification of the teeth generally, see the author's "An Introduction to Dental Anatomy and Physiology: Descriptive and Applied," 1913.

feature. This occurs chiefly at the sites of the excavations or sulci, though it is certain that the thickness of the tissue generally is not so great as usual. The surface is smooth and rounded.

Under higher magnifications the most internal stained areas are seen to consist of masses of coarse granules, the rods individually being ill-formed and structureless. The brown patches at the periphery of the enamel, however, show that the striæ resident in the rods are very pigmented, being stained brown (Fig. 5), while the intervals between the striæ shine with a bright lustre. But on
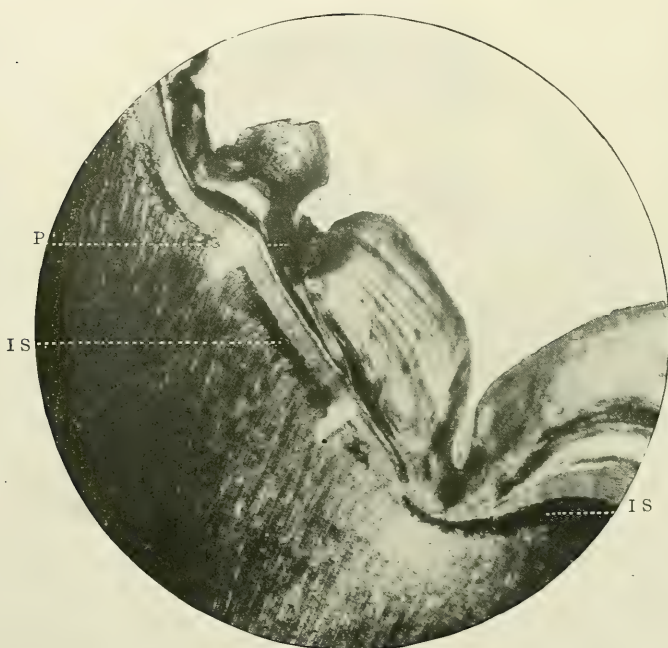
FIG. 3.—Similar to Fig. 1. Magnified 45 times. P. Pigmented enamel; I.S. Interglobular spaces in the dentine.

the whole the rods are not specially stained. The inter-columnar cement substance is clear and homogeneous. At the free margin the rods again become opaque and devoid of any histological elements.

The amelo-dentinal junction is sharply differentiated, and consists, as in normal conditions, of a linear series of myriads of convexities, each of which looks towards the dentine. The boundary is often crossed by many tubes and well-organised enamel spindles.

A little distance below the dentinal surface rows of unusually large interglobular spaces may appear (Fig. 7). They correspond in position to the fissures of the surface. There may be one, two, or even three rows of the spaces. In most cases they are very long and very broad. They are the usual, but not necessarily constant concomitant of enamel hypoplasia.

The dentinal tubules have apparently undergone no morphological alteration. The patho-histology of Nasmyth's membrane over
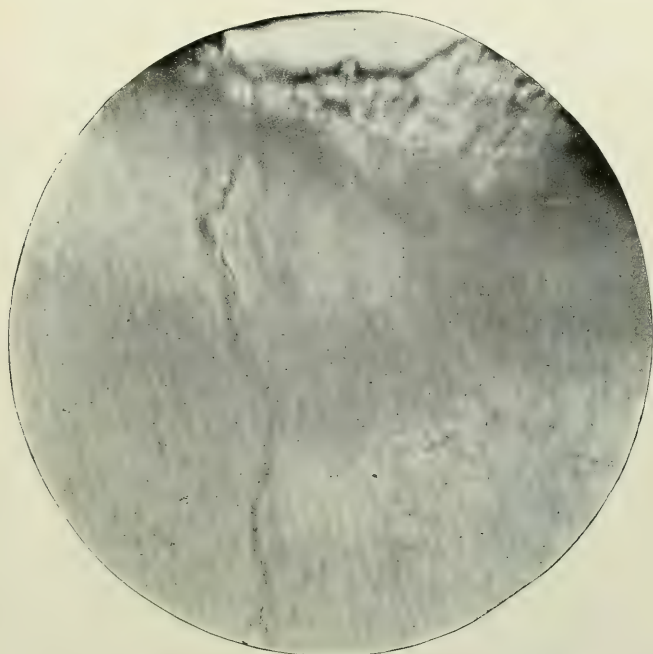
Fig. 4.—The same as the preceding. Magnified 750 times. Shows the faulty, irregular character of the enamel rods, and the pigmentation of a faint "interruption line."

hypoplasic enamel has never been fully investigated; but, according to J. G. Turner (*Trans. Odonto. Soc.* 1916) it may be considerably thicker than normal.

(ii) *Enamel Nodules*

Definition.—Small, solid, rounded, sessile bodies with white, shiny, smooth surfaces, situated below the necks of maxillary or
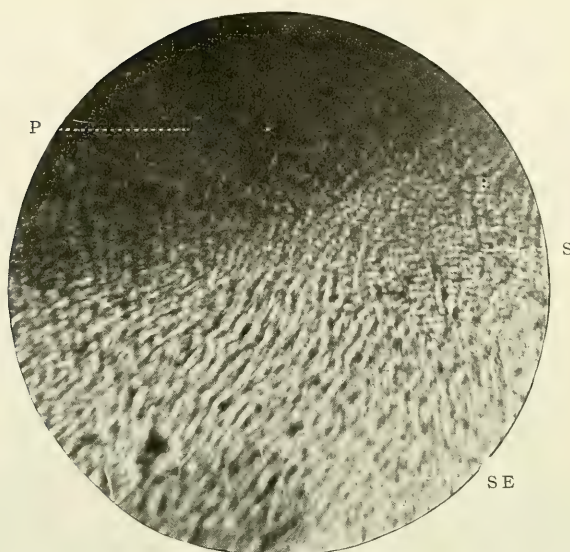
FIG. 5.—Hypoplasic enamel. Ground thin. Unstained. Magnified 350 times. Shows the prominence of the striæ of the rods through pigmentation. P. Patch of pigmentation; S. Marked striation of rods; S.E. Structureless enamel.
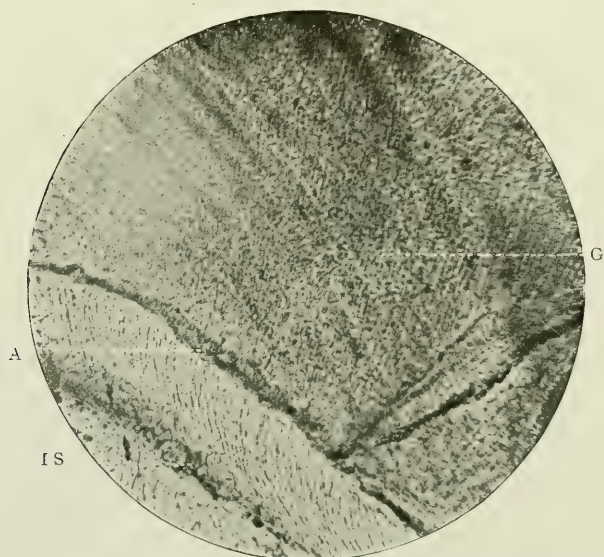
FIG. 6.—Similar to the preceding. Magnified 250 times. G. Granular structure of the enamel rods; A. Amelo-dentinal junction; I.S. Interglobular spaces.

mandibular molars with the dentine and cementum of which they are intimately connected. They have been classified[1] under the heading of composite odontomes. They are but rarely associated with the mandibular teeth.

Etiology.—They are produced by the local development and calcification of an aberrant prolongation of a portion of an enamel organ, which, while the rest of the tooth-band has become atrophied
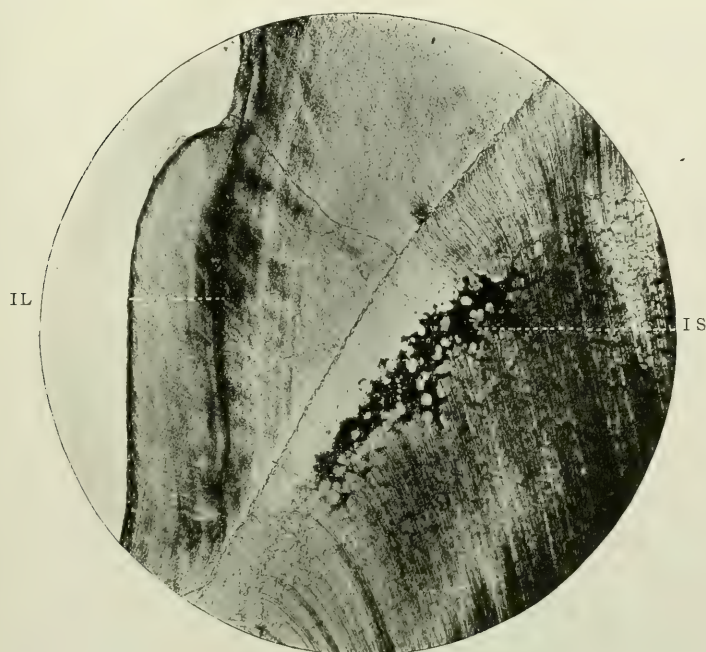
FIG. 7.—Similar to the preceding. Prepared by grinding, and photographed immediately after mounting. Unstained. Magnified 40 times. I.L. "Interruption lines;" I.S. Interglobular spaces.

and non-existent, persists in the region of the dental capsule, and after depositing lime salts disappears.

Macroscopical Appearances.—These little neoplasms bear a remarkable resemblance in shape, colour and lustre to white pearls. They present a marked contrast to the dull surface on which they are located (Fig. 8).

Secondary Changes.—As far as is known, they are unaffected by disease or retrogressive changes.

[1] "The Report on Odontomes" by the Committee appointed by the British Dental Association, 1914.

HISTOLOGY

A nodule is a hard mass, apparently spherical, but in reality hemispherical or semi-lunar in shape, consisting of a thick tubercle of pigmented enamel, whose constituent parts are very feebly matured. The rods are chiefly granular and homogeneous, and their transverse striæ scarcely visible. The striæ of Retzius and lines of Schreger may occasionally be found. The outermost surface is deeply coloured brown, the difference in the depth and gradations

FIG. 8.—Four maxillary molars showing typical enamel nodules.

of the stain being due, apparently, to the coarseness or fineness of the granules.

The free edge of the nodule is smooth and mammillated, while the attached margin is crescentic in outline and firmly adherent to a button of fine-tubed dentine, which, in itself, may exhibit structural defects, in the form of interglobular spaces. Enamel spindles and interglobular spaces, when they do occur, are filled with amorphous granular material. The crescents of the amelo-dentinal junction are not sharp, and many tubes cross the boundary.

FIG. 9.—Maxillary molar showing two enamel nodules.

Very rarely, enamel nodules, with the pathological appearances just noted, are found imbedded in the periphery of the dentine of teeth (see Fig. 12). In these instances they preserve their crescentic outlines, the difference being that the dentine does not project from the surfaces in the form of a tubercle or cone as is usually the case; though a cusp of dentine with radiating tubules does occupy the concavity of the nodule.

Nasmyth's membrane probably does not exist as such on the exposed surface of the nodule.
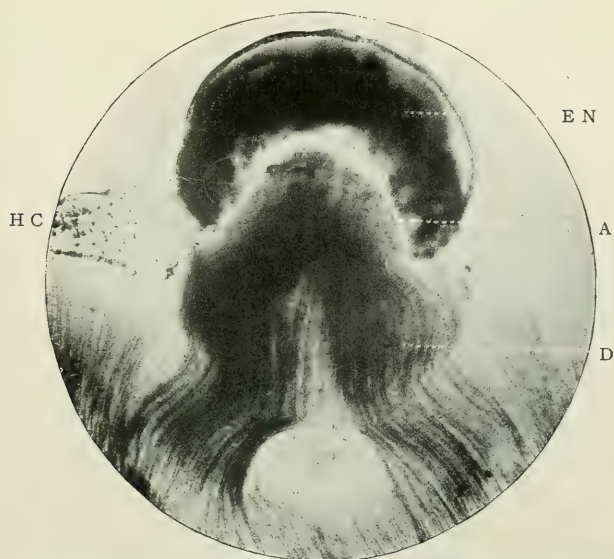
FIG. 10.—Vertical section of an enamel nodule. Prepared by grinding. Unstained. Magnified 20 times. E.N. Enamel nodule; D. Dentine; A. Amelodentinal junction; H.C. Hyperplasic cementum.
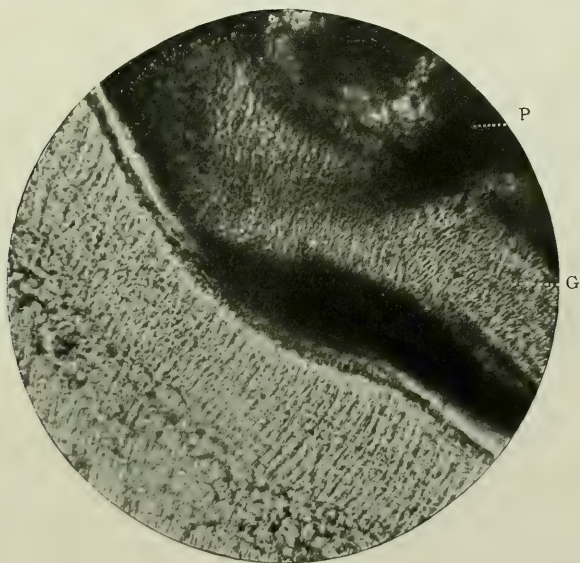
FIG. 11.—A portion of the preceding section. Magnified 250 times. G. Granular characters of the enamel rods; P. Dense pigmentation of the enamel rods, totally obscuring their structure.

(iii) *Pathological Pigmentation*

This, which is a natural condition in the enamel of some of the families of *Rodentia*, *e.g.*, Beaver, Coypu, or certain *Cetacea*, as in the persistently-growing incisor (tusk) of the Narwal, is seldom observed in human enamel. It must be distinguished from the green deposits constantly observed on the labial or buccal surfaces

Fig. 12.—An enamel nodule surrounded by hard tissues. Magnified 10 times. E.N. Enamel nodule; D. Dentine; F.D. Structureless dentine; C. Cementum; D.E. Detritus produced by grinding. (*Section prepared by Douglas Gabell.*)

of the teeth of young children, due to the agencies of chromogenic bacteria, which probably primarily affect Nasmyth's membrane.

Definition.—A brownish discoloration of the enamel of teeth.

Etiology is unknown. The tissue changes most probably have their origin as a congenital defect.

Macroscopical Appearances.—The enamel is bright and polished, but is stained a deep brown or yellow colour.

Secondary Changes.—None.

HISTOLOGY

There is little to be said about the microscopical structure of this condition. High powers reveal the fact that there are no imbrication lines; but the terminations of the enamel rods seem to be
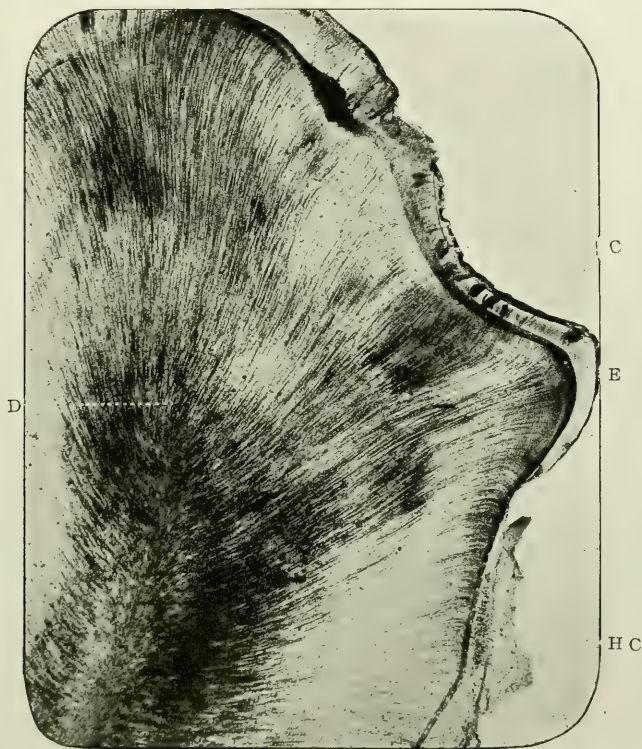
FIG. 13.—Vertical section of molar having nanoid enamel. Prepared by grinding. Unstained. Magnified 15 times. E. Enamel; C. Clefts; D. Dentine; H.C. Hyperplasic cementum.

covered with an opaque structureless narrow band of hard material, which is slightly laminated in a direction at right angles to the rods. Groups of rods running in a centrifugal direction possess yellowish striæ, with yellowish cementing substance between. In thin sections the colour is scarcely visible, but thicker masses of the enamel reveal the staining very well.

In the sections examined by the author there were no striæ of Retzius or Schreger's lines or enamel spindles; and the tissue itself was not particularly structurally defective.

(iv) *Nanoid or Dwarfed Enamel*

Specimens of human teeth apparently exhibiting absence of enamel or partial suppression of this tissue are occasionally met with. In no case however, investigated by the author, has there been entire absence of enamel, though in each it is greatly attenuated and stunted in growth.
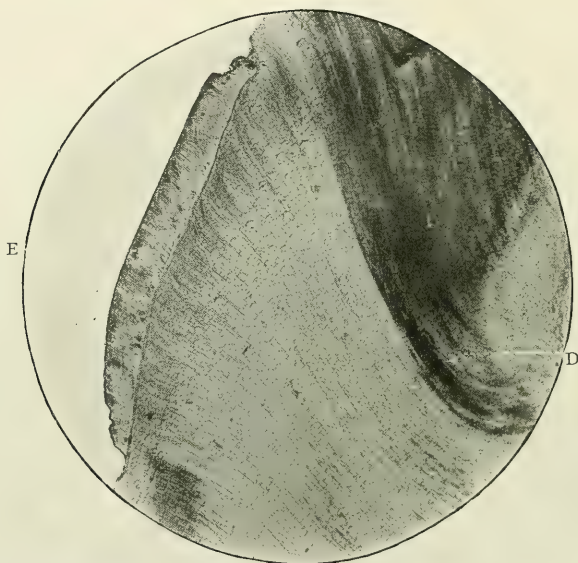
FIG. 14.—Similar to the preceding. Magnified 35 times.   E. Nanoid enamel; D. Dentine.

Definition.—Loss of normal thickness of enamel, producing a dwarfed condition, probably from failure of nutrition of the enamel organ, with the consequent production of its premature and retarded growth.

Etiology.—Perverted or loss of function of the ameloblasts.

Secondary Changes.—Caries and fracture.

Macroscopical Appearances.—The crowns of the teeth, which may themselves be fully developed, present rough lustreless surfaces, stained somewhat a slight yellow colour. The probability that the chromogenic bacteria of the mouth, or organic post-mortem

changes in the tissues have produced the staining, is very remote. In one unique instance, three of the four third molars in the same mouth shewed signs of loss of enamel immediately after eruption, and it is equally certain that caries was absent.   In a tooth in the

FIG. 15.—A section through the jaw of a young ricketty monkey during development, the deciduous and permanent teeth being *in situ*.   Prepared by Weil's process.   Magnified 20 times.   E. Enamel of permanent tooth exhibiting the structural defects described in the text; D. Its dentine; P. Its pulp; M. Dentine of deciduous tooth.   (*The section was prepared by Sidney Spokes.*)

possession of the author, one tiny mound of enamel was observed by the naked eye; while, on microscopical examination, two very minute masses of enamel were discovered over one of the cusps of this anomalous tooth, which in itself was exceedingly dwarfed and misshapen.

2

HISTOLOGY

The striæ of the enamel rods are very conspicuous, and the inter-columnar substance marked. A tinge of yellow pigmentation is discernible in places. Numerous microscopic clefts pass in from the free surface of the tooth for varying distances. These are not due to manipulative processes consequent on the act of making the section.

The dentine is, as a rule, normal (Fig. 13); though in the last-named specimen, interglobular spaces and vascular canals were found in places.
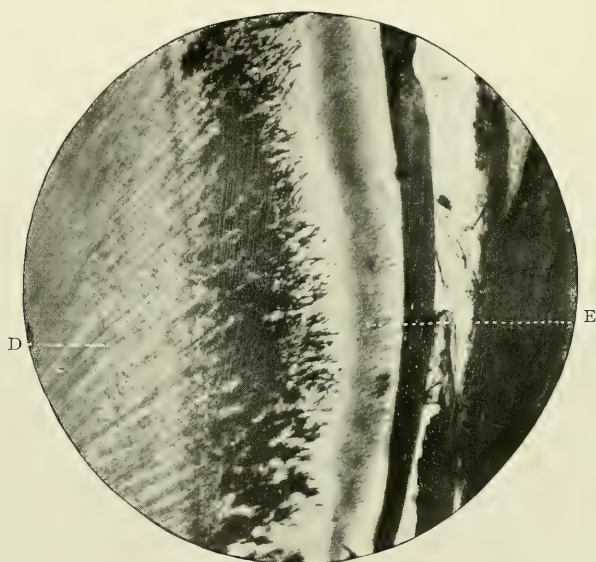
FIG. 16.—A portion of the preceding. Magnified 50 times. E. Enamel; D. Dentine.

(v) *Rachitic Enamel*

Little is known of this condition: but sections lent to the writer by Mr. Sidney Spokes, and photographed (Figs. 15 and 16) show that during development, certain large, numerous clefts possibly containing, in the recent state, soft material, can sometimes be observed on the inner aspect of the tissue. Measurements of the length of the longest of the spaces in this particular specimen showed them to be 0.5 mm. The photomicrograph, taken by the author

from the original preparation, are of the parts in and around the evolving permanent tooth of a monkey affected by rickets, and are most likely perfectly similar to those obtaining in like circumstances in man.

Fig. 17.—A section of a human syphilitic incisor. Magnified 175 times. It shows extremely imperfect enamel, the spaces of which do not contain living material. E. Structurally defective enamel; I.S. Interglobular spaces in dentine. (*The section was prepared and photographed by Leon Williams.*)

The enamel of the corresponding deciduous tooth was likewise affected, but in a slighter degree.

(vi) *Syphilitic Affections*

Definition.—Certain misshapen, deciduous and permanent teeth associated with congenital and inherited syphilis.

Synonyms.—"Pegged teeth," "Hutchinsonian teeth."

Etiology.—They are the result of stomatitis, a secondary syphilitic lesion inherited by children, in which the influence of the inflammation on the cells of the enamel organ and dentine papilla is permanently recorded in the surface and shape of the incisors, canines and first molars.

The *Spirochæta pallida*, according to Cavallaro of the University of Florence, "Nouvelles Observations sur la Dentition chez les Syphilitiques Héréditaires," 1909, can be demonstrated in the dental capsule, and in the pulp on the walls and in the neighbourhood of the blood-vessels.
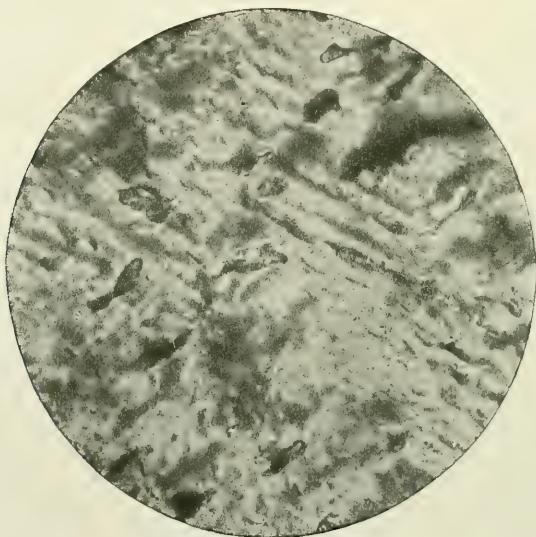
FIG. 18.—A portion of the enamel from the preceding section. Magnified 600 times. It shows lacuna-like spaces found in syphilitic enamel. (*Photomicrograph by Leon Williams.*)

In the same year, also, this organism was observed in the dental tissues by Professor Pasini of Milan, and Assistant Professor Lombardo of Modena.

Macroscopical Appearances.—The maxillary incisors are most commonly affected, and present characteristic and unique appearances. These are (i) a slight notch or groove, crescentic in shape, on the cutting edge; and (ii) smallness of the teeth themselves, which have convex sides and rounded-off angles.

The mandibular incisors are usually conical and small.

Secondary Changes.—None.

HISTOLOGY

The surface is not absolutely smooth, but "contains pits which are filled with micro-organisms of a reddish-brown colour, thus indicating that caries is not in progress. The enamel rods are poorly calcified; large lacuna-like spaces, which appear to be filled with organic matter, are seen, some of which have radiating proc-
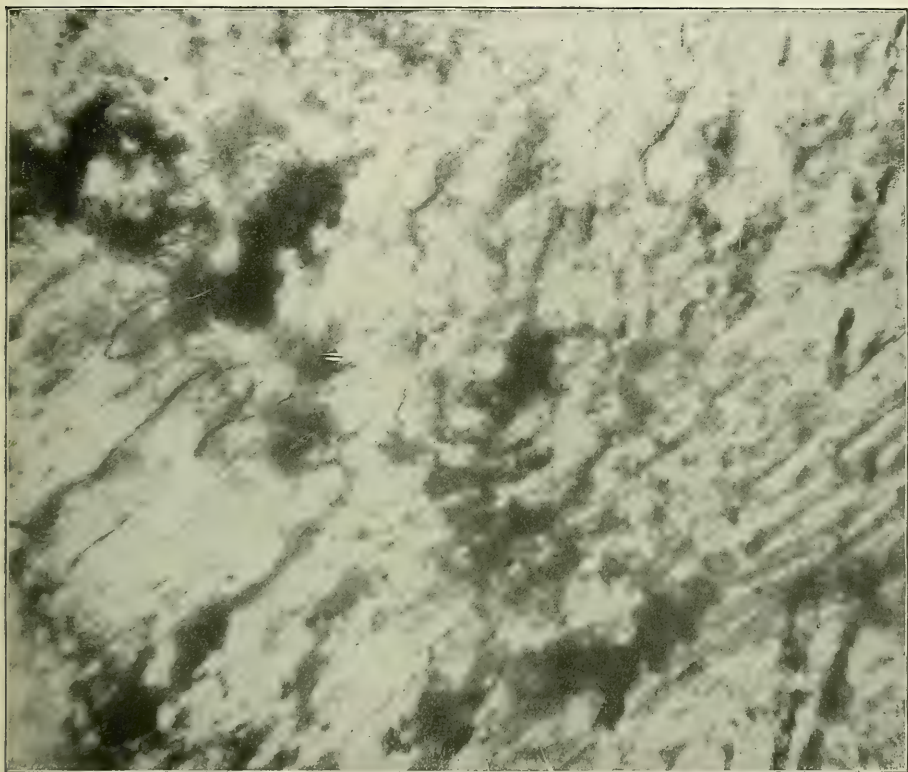
Fig. 19.—Another portion from the same section.   Magnified 800 times.
(*Photomicrograph by Leon Williams.*)

esses, while others communicate with the dentine by means of canals." (Leon Williams.) Other portions exhibit large cavities of various shapes. The dentine contains interglobular spaces (see Figs. 17 and 20).

B.—ACQUIRED DISEASES

The morbid affections of the enamel which have been acquired since its full maturity has been reached are of interest, because,

as a rule, they are inseparable from certain lesions of the neigh-
bouring dentine and cementum. The dental pulp, entirely un-
affected by the retrograde changes which have taken place in the
development of the teeth, too, often suffers. Naturally also, local
conditions play a great part in the immediate causation of this
group of acquired diseases, although erosion probably of all enamel
lesions may be predisposed to more by general systemic dyscrasia
than by inflammatory conditions or physical disturbances of the
mouth.

<div align="center">(vii) Abrasion</div>

Definition.—A term applied to the rapid wasting and destruction
of enamel and dentine by friction set up by foreign bodies.
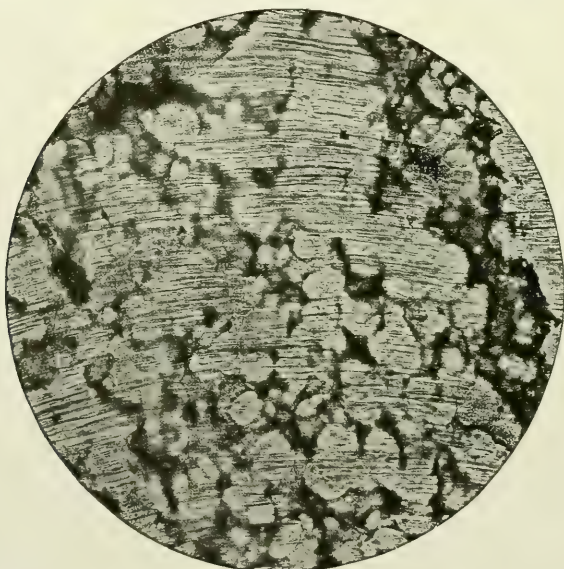
FIG. 20.—A portion of the dentine from the preceding section. Magnified
250 times. It shows the interglobular spaces of syphilitic dentine. (*Photomicro-
graph by Leon Williams.*)

Etiology.—Injury by mechanical appliances around the teeth and
friction from careless or excessive use of the tooth brush are said to
be potent factors in its causation.

Macroscopical Appearances.—The cavities on the surface of the
crown are very flat, dull, rough and superficial, being stained some-
what yellow. The outline is usually ovoid. Hyperæsthesia may
or may not be present.

Secondary Changes.—Caries, fracture, secondary dentine in the pulp chamber.

HISTOLOGY

Early stages show a breach of surface at the cervical region of the teeth, with loss of the thin edge of enamel and cementum. Pigmentation is present. The dentinal tubes appear similar to those of attrition. There is this marked difference, however, between the two, *viz.*, caries may proceed more or less rapidly in the former, and not at all in the latter. Later developments lead to softening of the dentine matrix, deeper invasion of micro-organisms, and destruction of the hard parts around the cavity.

(viii) *Absorption of Enamel*

There are two kinds of absorption of enamel—(α) external and (β) internal. Both rare, the former may be, in its earliest stages, unassociated with absorption or any morbid change in the dentine, —though at times it may accompany absorption of both dentine and cementum—and proceeds from without inwards; while the latter is wholly an occasional accompaniment or corollary of the phenomena of dental caries, and proceeds from within outwards.

Definition.—That loss of enamel substance after the tissues have been fully completed, which is due to pathological and not physical or physiological or mechanical causes.

(α) *External Absorption*

Etiology.—Probably sometimes the result of (α) acid solutions coming into immediate contact with the surface of enamel when there has been some localised suppuration: and sometimes (β) the effect on the enamel surface of the functional activities of osteoclasts. The anomaly, which is rare, can be observed in connection with the retarded eruption of teeth in apparently edentulous mouths, where inflammation of the soft parts in the neighbourhood of the tooth has been induced by pressure from artificial dentures. In certain cases brought under the immediate notice of the author (a) malplaced canines undergoing moliminous eruption in an inverted position had been the cause in two instances; and in a third (b) an unerupted third mandibular molar was the affected tooth.

Macroscopical Appearances.—As one type, the following description may be given of one of these specimens.[1] The crown, retaining

[1] See "A Case of Retarded Eruption," by Warburton Brown. *Journal Brit. Dental Association,* June, 1900.

in some measure its original conical form, was deeply hollowed out by means of curious excavations, some of which penetrated the pulp cavity (see Fig. 21). The surface was pitted and brown in colour. Its cervical measurement was 22 mm., and a large cavity 13 mm. in length, extended round the labial and distal surfaces when the tooth was viewed in its normal position. The enamel and dentine, as a thin shell, were complete over half the labial and half the mesial surface.

The crown was greatly pitted, having a deep fissure in the distal surface, as is seen in the photograph (Fig. 22). It was attached to the neck of the tooth, mainly on its lingual aspect—(Fig. 23)—as well as by means of a column of dentine in the central axis of the

Fig. 21.   Fig. 22.   Fig. 23.

Fig. 21.—The labial aspect of the unerupted tooth described in the text: Fig. 22.—Its distal surface; Fig. 23.—Its lingual side. Note.—The surfaces of this tooth are named, as if the canine had finally erupted in a normal manner, and assumed a normal position in the dental arch. A. Deep excavation; B. Absorption extending to the pulp cavity, the side of which is seen at C.

tooth, which occupied what was probably the original pulp chamber.

The total area of enamel present was about 2 mm. square.

The tooth generally was normal in size, yellowish in colour (indicating age), and had some slight amount of hyperplasia of the cementum over its apical region.

Secondary Changes.—None usually; but, exceedingly rarely, deposition of bone.

HISTOLOGY

In (*a*) cases the minute anatomy is interesting, as Howship's foveolæ, almost universally associated with absorption of the hard tissues of teeth or bone, are entirely wanting. The outlines of the cavity are irregularly flat or oval, possessing none of the round bay-like recesses seen in absorption in other tissues. Although these absorption-areas appear to be wanting, it would be difficult

to explain the presence of such large excavations in the hard parts, if the question of osteoclastic activity were put entirely out of court. Hence it would seem reasonable to suppose that the greater proportion of the spaces had been produced by the functions of these cells.
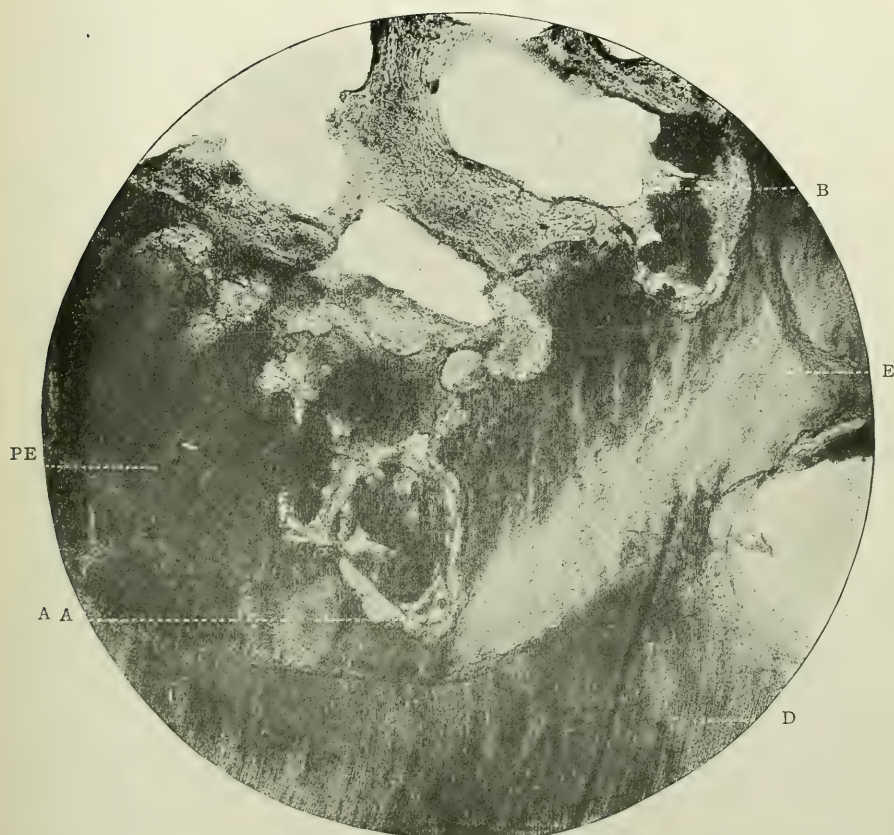
FIG. 24.—External absorption of enamel. Prepared by grinding, and stained by impregnation with coloured collodion. Magnified 40 times. E. Enamel of normal structure; P.E. Pigmented enamel; A.A. Absorption areas filled with osseous material; B. Trabeculæ of compact bone; D. Dentine.

The enamel rods appear as if broken off, and at the extreme edge there is a slight dissolution of their intercolumnar cement substance. There may be, or there may not be, some pigmentation of the parts.

It is extreme conditions, and such as that already cited in full, which are accompanied by absorption of dentine.

In the (β) case of a specimen, probably quite unique, for which

the author acknowledges his indebtedness to Sir Francis Farmer, re-
markable absorption had occurred, and had been followed by a
most unmistakable deposition of cancellous bone (see Fig. 24).
Here the foveolæ of Howship are clearly distinguished, loss of enamel
having in some parts extended almost to the amelo-dentinal junc-
tion. Osseous material, easily recognised macroscopically as well
as by the microscope, occupied the excavations and practically
restored the greater part of the absorbed crown of the tooth to its
normal height. The history of the tooth in no wise suggested
this interesting histological abnormality.

(β) *Internal Absorption*

Etiology.—Caries of dentine.

Secondary Changes.—Deposition of calcified material (osteo-
dentine) may occur, as in the case recorded by Mr. J. A. Woods,
"A case of Absorption," *Journal British Dental Association*, April,
1902, pp. 193–197 (see Figs. 25, 26).

HISTOLOGY

In a letter to the author Mr. Woods describes the condition as
follows:—

"Mr. F. Rose, at a meeting of the British Dental Association,
showed a partly absorbed maxillary third molar which he had re-
cently removed. It came away without using any force and prac-
tically left no socket, and only a very shallow crown. On naked eye
examination it appeared to be merely the crown of a tooth, and to
consist mainly of the enamel filled with a somewhat soft calcareous
mass. He offered it for histological examination, and the report
made at the next meeting was as follows:—

"'On scratching the inner part a distinct grating noise could be
heard, which pointed to calcareous elements.

"'On proceeding to cut a section, I found it would be necessary
to make an ordinary ground one, as a decalcified section would not
show any of the soft tissues, which had already been destroyed
by drying, etc. It was not possible to get a very thin section without
running a great risk of destroying the part showing the new tissue,
which, of course, was the most important portion.

"'The accompanying photomicrographs are taken from various
parts of the one section and show most of the important points.

FIG. 25.—Internal absorption of enamel. A. Enamel; B. Normal dentine very full of interglobular spaces; C. Cavity in the dentine formed by absorption and then deposition of "osteo-dentine;" D. Enamel penetrated and absorbed and filled in with "osteo-dentine." (*Photomicrograph by J. A. Woods.*)
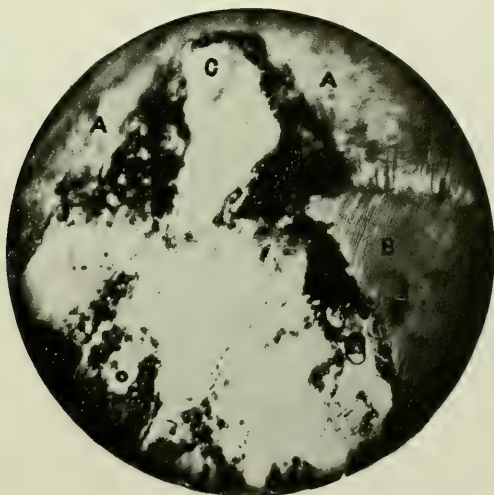
FIG. 26.—A higher magnification of the preceding, showing absorption of the dentine and enamel from the pulp surface, and deposition of "osteo-dentine." A. Enamel; B. Normal dentine; C. Deposition of "osteo-dentine" (?) in enamel. (*Photomicrograph by J. A. Woods.*)

"'A low magnification showed that a large portion of the normal dentine had undergone absorption, and that in one part (a cusp of the molar) the inner surface of the enamel had been removed. All this normal tissue has been replaced by a mass of cancellous calcareous tissue which can probably be best termed osteo-dentine.

FIG. 27.—Vertical section of molar showing internal and external absorption of enamel and dentine. Prepared by the Koch-Weil process. Stained with Grenacher's alcoholic borax-carmine. Magnified 45 times. D. Primary dentine; B. Compact but irregularly formed bone filling up pulp cavity; A. Compact bone filling up absorption area in primary dentine; C.B. Bridge of compact bone crossing over areas of internal and external absorption.

"'Fig. 25 is a rather higher magnification of the same part. It will be observed that the dentine contains a large number of interglobular spaces; this, however, is not by any means uncommon

in third molars.   It will also be observed that in several places the absorption has penetrated the dentine in different places, and so one section gives the appearance of several isolated patches of absorption.

"'The deposited material consists of spicules as seen in cancellous bone, the spaces being probably filled in the recent condition with pulp matter.   The presence of lacunæ can be seen in various parts of the osteo-dentine.

"'Fig. 26 gives a clearer view of the absorption of the dentinal surface of the enamel with the new material *in situ*.

"'Another portion of the section shows a patch of cementum, part of which has been removed by absorption and osteo-dentine has taken its place.'"

(ix) *Attrition*

Definition.—Gradual wearing away of the hard parts through the physical and physiological agencies of mastication of food.

Etiology.—A constant accompaniment of senility, it is probable that in the young it may occur under the influence of certain mechanical forces, the result of imperfect occlusion.   In the former it is a general condition, in the latter localised.

Macroscopical Appearances.—The morsal surfaces of molars present a bright polished flattened table-land of hard tissue.   Anterior teeth may be worn away so that half the crown of the tooth may have disappeared (see Fig. 28).   Tartar is often present over the roots of these teeth.

Secondary Changes.—Fracture, formation of secondary dentine in the pulp.

HISTOLOGY

The enamel is very pigmented, and the rods are cleanly cut at right or acute angles to their courses.   The primary dentine is free from caries, but slightly coloured in patches which run throughout its entire thickness.   The dentine is cut transversely or tangentially, sometimes at right angles to the tubules, at others parallel with them.   The cut is sharp, and looks as if made by a keen razor. Interglobular spaces are frequently present in greater numbers than usual.

The secondary dentine is well formed and fine tubed, and fills most thoroughly the coronal and cervical regions of the former pulp cavity.

(x) *Erosion*

Definition.—Progressive destruction of the exposed surfaces of
teeth, producing cavities which are peculiarly dense and polished,
and in the majority of instances hypersensitive on receiving tactile
impressions.

Synonym.—"Cuneiform defects."
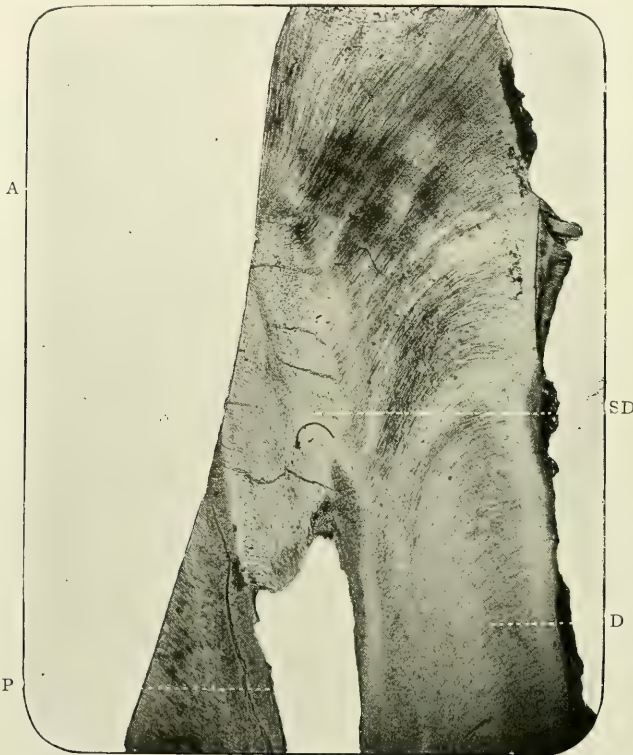
FIG. 28.—Sagittal section of a human incisor, showing marked attrition of
the crown.   Prepared by grinding.   Unstained.   Magnified 12 times.   A. Worn
surface of the tooth; D. Dentine; S.D. Secondary dentine; P. Pulp cavity.

Etiology.—Little is known as to the origin of this common con-
dition which is found in the teeth of man the wide world over.   Much
has been written on the subject, and many experiments to induce
it artificially have been performed.

In England the lesion is usually ascribed to gout or allied diseases,
affecting the small mucous glands of the lips or gums, by setting

up a state of local congestion, and causing these bodies to pour out an acid instead of an alkaline or neutral secretion. This acid mixture attacks the enamel or dentine or cementum of teeth at their cervical margins, and decalcifies those tissues.

It is exceedingly probable, however, that the condition is predisposed to by the denudation of dentine at the necks of teeth by the thin edges of enamel and cementum which, as a rule, overlap it, when—in other words—these two tissues do not meet *bout à bout* (see Chap. III, Vol. I).

The action on or morbid changes in Nasmyth's membrane are entirely unknown. Tomes[1] attributes the affection to a chemical solution of the tooth from acids generated by the fermentation of mucus, or this material affording a suitable nidus for acid fermentation. Salter combines abrasion and erosion under the inclusive title of "Surface wear," and he divides the causes into—

(α) Predisposing—inherent softness of structure, as in syphilitic teeth:

(β) Exciting—molar-mastication by incisors, gritty food, hard tooth-brush, and friction from wearing a denture.

On the Continent of Europe, six chief views are held:—

1. *The Chemico-mechanical Theory*, supported by Bastyr, Brandt, Sheff, Shlenker, and Walkhoff, in which an exceedingly thin layer of dentine is decalcified by the constant influence of weak acids generated in the mouth, and finally removed by mechanical means, such as tooth-brush, powders, etc.;

2. *The Mechanical Theory*, due to attrition by friction of food, etc., advocated by Parreidt and Niemeyer;

3. *The Chemical Theory* of Baume;

4. *Caries* by Leber and Rottenstein, Magitot,[2] etc.;

5. *The Exfoliation Theory* of Baume, which maintains the hypothesis that the superficial layers of the dentine not covered by gum or enamel "die," and fall off mechanically, the polishing of the denuded spaces being brought about by the rubbing of the lips, etc.;

6. *The Dechondration or Decalcification of the Dentine Theory* of Znamensky.[3] According to this careful observer enamel

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[1] Tomes & Nowell: "A System of Dental Surgery," 1906.

[2] "Recherches sur la Carie des Dents, Paris, 1871."

[3] "On the question of the origin of the Cuneiform Defects of Teeth." *Journal Brit. Dent. Assoc.*, pp. 8 *et seq.*, 1898.

plays no part in the process; but there is mechanical removal of isolated particles of lime salts in the eroded regions. This is due to "unequal abstraction of the 'gelatine-yielding' substances of the dentine," and the subsequent swelling of its organic constituents.

Erosion sulci have been said to have been found on human teeth worn on a denture as substitute in case of loss, and also in ovarian teeth (see page 467).

Macroscopical Appearances.—The cavity of erosion is a sharp-edged, smooth, highly polished groove at the necks of teeth, running transversely to their axes. *In position*, these acquired defects are most often seen on the labial or buccal aspects, and only very rarely over the lingual or palatal sides of the teeth. *In outline* they are

A   B
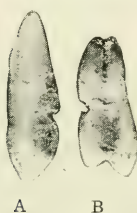
FIG. 29.—Maxillary left canine, and maxillary left first premolar, from mouth of a man aged 54, showing erosion cavities on the *palatal* surfaces. A. Distal aspect of canine. B. Mesial aspect of premolar.

more or less wedge-shaped, with edges sharply cut and well-defined. Their surfaces are smooth, bright and polished; by means of a hand lens, saucer-like cavities may sometimes be discerned. If these tiny subsidiary excavations or spaces exist in great numbers, they may impart a dull, even rough, surface to the floor and walls of the grooves, which then may be stained any colour, from yellow to brown, black, or even green. In deep grooving the enamel is undermined. *In number*, as a rule, one large file-like cut is seen, but occasionally several exist on the cervical region of the teeth, especially when a large portion is denuded.

HISTOLOGY

The earliest evidence of change, according to Baume, is the presence of cup-shaped depressions analogous to Howship's foveolæ, covering the eroded surface, which is now uneven. Fissures in the neighbouring dentine are simultaneously produced. Further internally placed, is a band of translucent dentine.

Underwood has noticed (*Journal Brit. Dent. Association*, Vol. xix., pp. 470–2) a hitherto undescribed phenomenon connected with enamel the subject of erosion. This is the presence of exceedingly tiny interglobular spaces, with accompanying calcospherites (see photographs, Figs. 31 and 32). These spaces are apparently scattered throughout the tissue. The writer adds that the condition
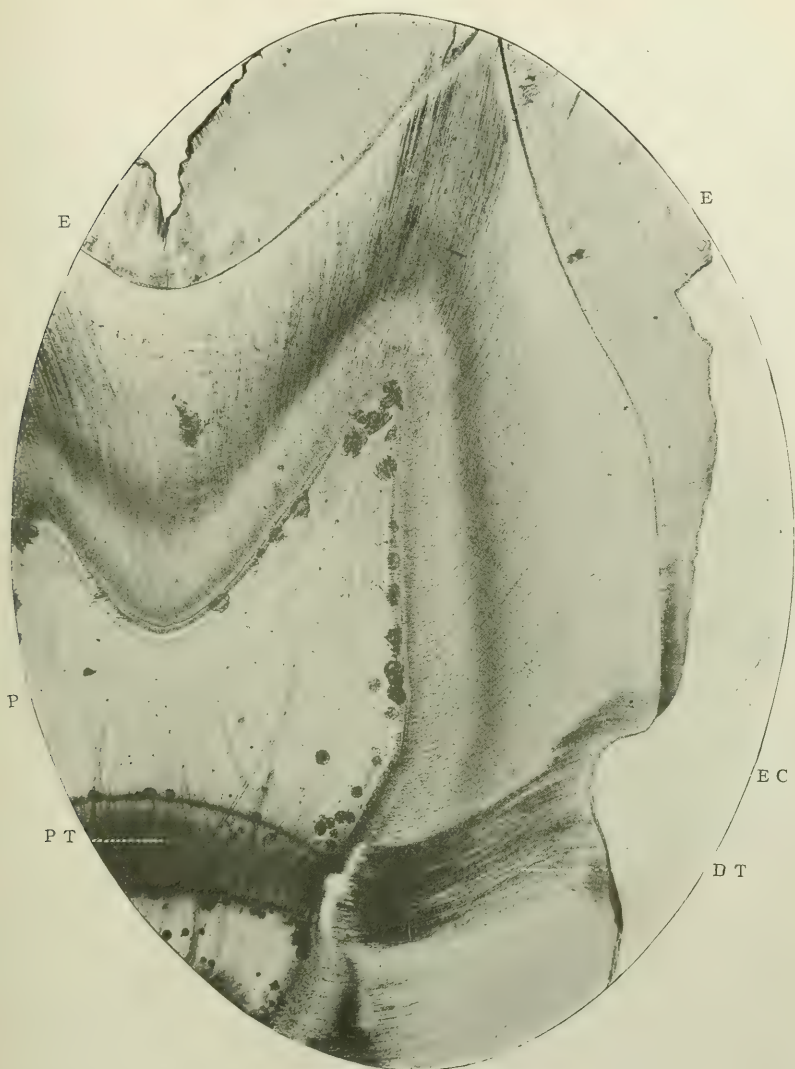
FIG. 30.—Coronal section of a human molar. Prepared by Weil's process. Magnified 15 times. E.C. Erosion cavity on buccal side of the tooth; D.T. Dentinal tubes which are probably completely calcified; E. Enamel; P. Pulp; P.T. Band of pulp tissue which has undergone some degenerative (?) changes. The small black masses in the pulp are probably clusters of borax crystals, deposited in this manner by the salts in Grenacher's alcoholic borax-carmine stain.

3

differs in every respect from the *so-called* erosion found in the teeth of *Otaria*, first described and figured by Murie in the *Trans. Odonto. Soc. of Great Britain* for 1870.

The granularity of the rods is exaggerated.

That the underlying dentine is affected in earlier stages of the disease is shown by the fact that the tubules are calcified throughout. That is to say, in sections of teeth prepared by the Koch-Weil process they refuse to stain, but maintain a dark black colouration through all their length (Fig. 30).

The pulp has deposited a layer of adventitious dentine opposite the breach of surface. This is truly Salter's "dentine of repair"
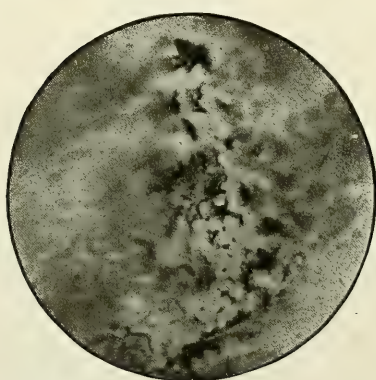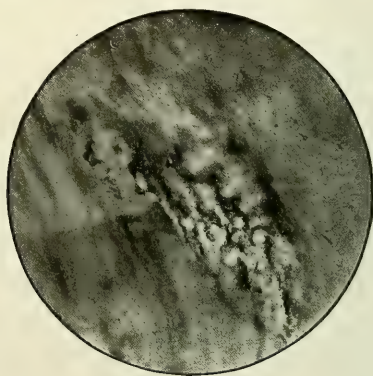
FIG. 31. FIG. 32.

FIG. 31.—Erosion of enamel. Prepared by grinding. Unstained. Magnified 750 times. Shows interglobular spaces and calcospherites. (*Section from the collection of A. S. Underwood.*) (*Photomicrograph by Andrew Pringle.*)
FIG. 32.—Similar to the preceding. From the same source. (*Photomicrograph by Andrew Pringle.*)

(see Fig. 34). Its structure is of an irregular, indefinite character. A well-organised system of tubes is wanting, though spaces of an unusual type may be found. In the photomicrograph (Fig. 35) the pulp exhibits a different pathological condition to that in the Figure on page 33. The specimen is interesting, showing, as it does very clearly, the clefts described by Baume, the "dentine of repair," and the pulp undergoing retrogressive or fibrous changes.

(xi) *Fungoid Excavation*

Definition.—The boring by fungoid organisms of large tubes in the hard parts of teeth.
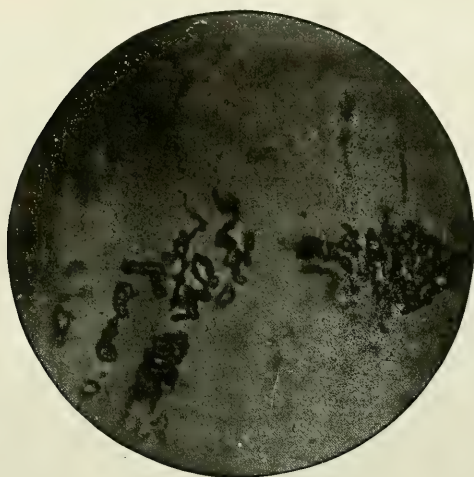
FIG. 33.—Appearances mentioned in the text in the enamel of the sea-lion. (*Photomicrograph by Andrew Pringle.*)

FIG. 34.—Erosion, showing deposit of "dentine of repair" on the internal aspect of the dentine. Magnified 40 times. E.C. Erosion cavity; S.D. Adventitious dentine. (*Section prepared by Douglas Caush.*)
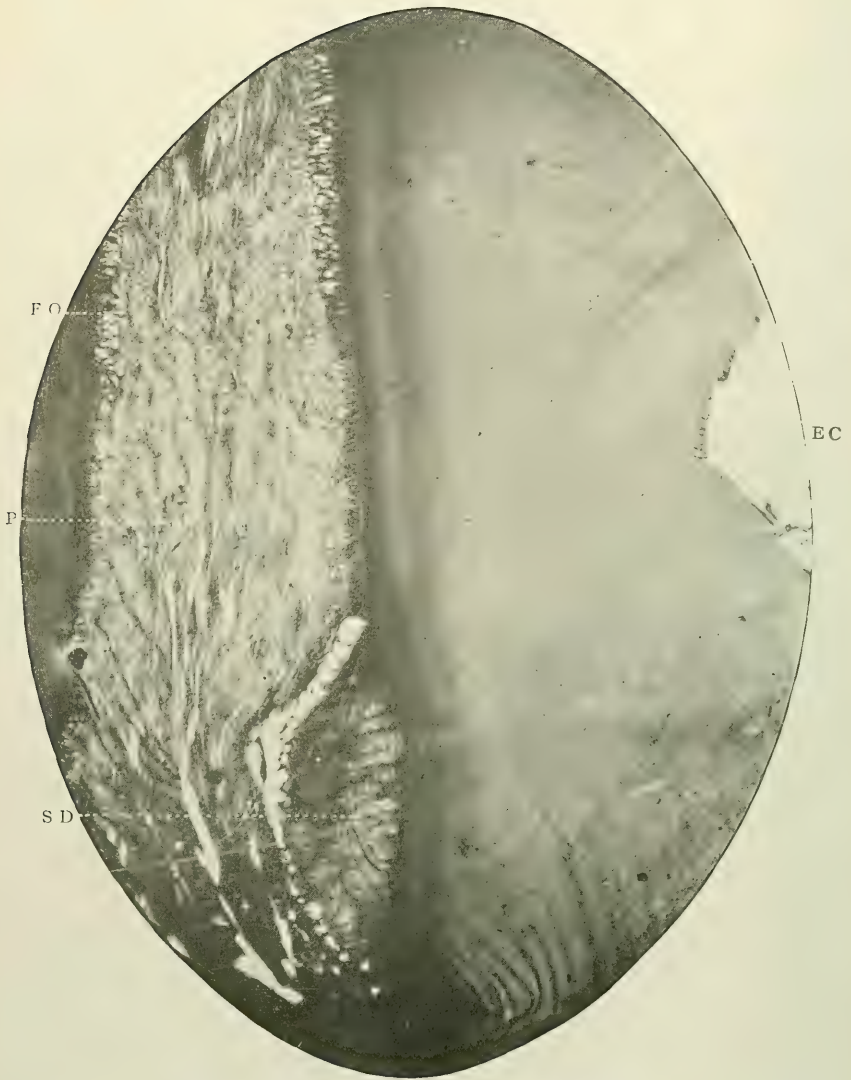
FIG. 35.—Sagittal section of human incisor. Stained with hæmatoxylene.
Magnified 45 times. E.C. Erosion cavity on surface of which can be seen
Baume's clefts; P. Pulp tissue undergoing degenerative changes; F.O. Atrophic
odontoblasts; S.D. Fibrillar adventitious dentine. (*Prepared by the Author's
process.*)

Etiology.—It is supposed to be due to the peptonising or acidifying effects of a *Saccharomyces* equally in the substance of the enamel, dentine, and cementum.   The condition is extremely rare.

Secondary Changes.—None.

<div align="center">HISTOLOGY</div>

Enamel, dentine, and cementum may suffer in different degrees. Two interesting cases have been recorded.   Tomes (*Trans. Odonto.*
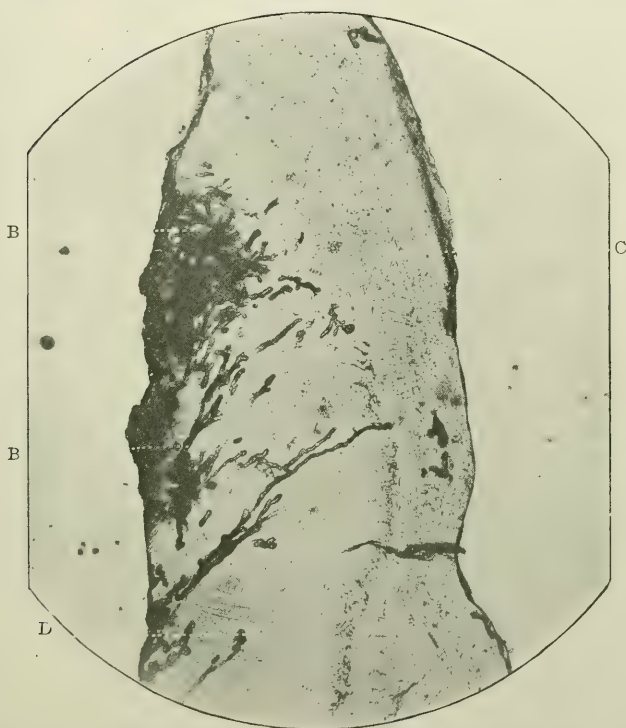
Fig. 36.—Ground section of dentine and cementum.   Unstained.   Magnified 20 times.   D. Dentine; B. Excavations made by the fungus; C. Cementum. (*From a specimen in the collection of Charles S. Tomes.*)   (*Photomicrograph by Douglas Gabell.*)

Soc. Vol. xxiv., pp. 90–91) describes the first, where a tooth picked up in an ancient graveyard had presumably been buried for a great number of years.

The dentine of the root was largely excavated in all directions by tunnels or channels of uniform diameter, which most probably were caused by a yeast, possibly one of the *Saccharomyces*.   "In

many places the borings followed two directions, more or less at right angles to one another—the one being along the dentinal tubes, the other along places corresponding to those incremental layers, along which disintegrating dentine so often breaks up. Where the dentinal tubes were traversed, the enlarged portion passed abruptly into that which was unchanged, so that the idea of a mere
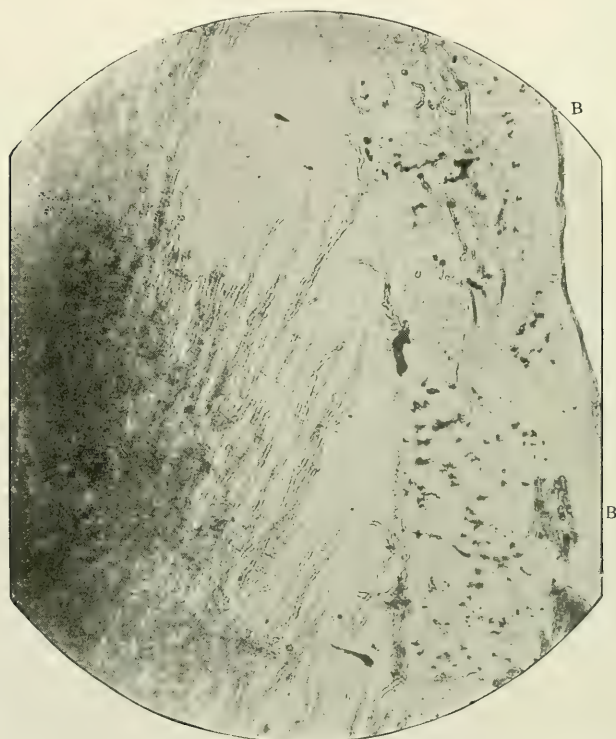
FIG. 37.—Similar to the preceding, and from the same source. Stained with carmine. Magnified 40 times. B. Borings in cementum. (*Photomicrograph by Douglas Gabell.*)

chemical solvent creeping down the tubes was negatived. And although its usual course was along lines which might be regarded as those of least resistance, it was perfectly capable of drilling the dentine in any direction whatever, as is seen in Fig. 36, in which the tunnelings were in all directions, quite irrespective of the tubes or the lines of growth."

"These sections prove that a fungus can, unaided or aided only

by the decomposition of the organic material present in dentine, drill freely in any direction."

Penetration of the cementum is clearly seen in Fig. 37.

The second instances are those recorded by Professor Duckworth, of the Physical Anthropology Laboratory at Cambridge University. (*Trans. Odonto. Soc. of Great Britain*, Feb., 1901, p. 89), in an article entitled "Some dental rudiments in Human Crania." In these
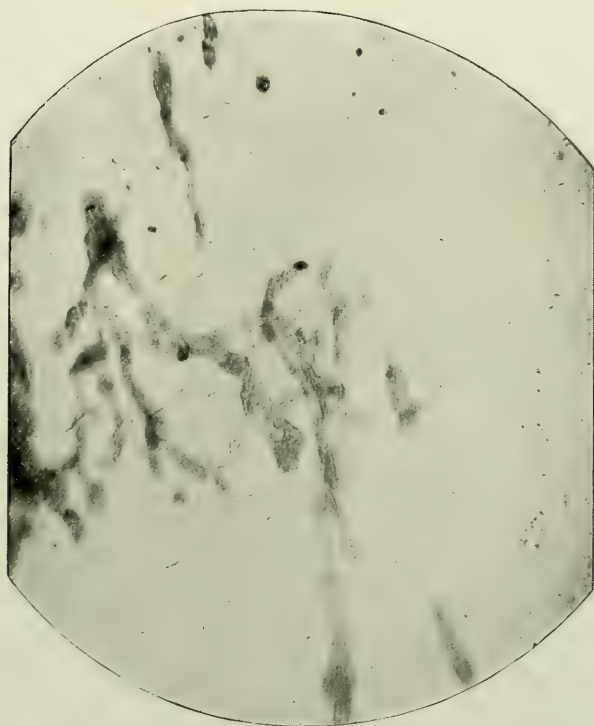
FIG. 38.—Similar to the preceding. Stained with carmine. Magnified 160 times. (*Photomicrograph by Douglas Gabell.*)

tiny calcareous fragments, which probably represented vestigial third premolars (similar to those of the New World Apes), true dental tissues were found, the enamel and cementum of which were channelled through and through with the borings made by one of the *Blastomycetes*.

The fragments came under the personal notice of the author, who in a microscopical report wrote: "Specimen No. 2154 (*e*)

Columns (probably enamel), chiefly seen in transverse section, though sometimes longitudinally cut. Not very brightly outlined, nor possessing marked striæ, nevertheless clear and unmistakable. Numerous large tubes or channels with bulbous or rounded extremities run here and there throughout the tissue." "Root portion of tooth. Dentinal tubes are marked and when filled with detritus from grinding very black. Cementum thick, few lacunæ

FIG. 39.—Channelling of the enamel. Prepared by grinding. Magnified 50 times. E. Enamel; C. Sinuous tube passing from without inwards to end at, A. The amelo-dentinal junction; D. Dentine. (*Photomicrograph by Douglas Gabell.*)

and canaliculi, but incremental lines very apparent. Traces here and there of fungoid burrowing. Specimen No. 2154 (*n*) Transverse section of dentine. Cementum practically structureless. Large channels produced by *Saccharomyces mycoderma*, confined, in this preparation to the cementum; granular layer marked."

Abnormal channelling of the enamel has been known to occur. In the specimen, from which Fig. 39 was reproduced, a long winding canal can be observed leading from the periphery of the tooth to the amelo-dentinal junction which it did not pierce. There is no history of the case. It is certainly a developmental error; but its method of formation is absolutely unknown.

CHAPTER II

THE PATHOLOGICAL CONDITIONS OF THE DENTINE

MICROSCOPICAL ELEMENTS FOUND IN:—(i) Dilaceration; (ii) Gemination; (iii) Developmental defects; (iv) Pigmentation; (v) Nanoid dentine; (vi) Vascular Canals; (vii) Absorption; (viii) Adventitious Dentines; (ix) Pathological Pigmentation; (x) Senile Dentine.

A.—DEVELOPMENTAL DISEASES

(i) *Dilaceration*

Definition.—A permanent malformation of the teeth, usually in their cervical or radicular portions, in which the hard parts are deviated from the axial straight line in such a way as to form an angle with it.

FIG. 40. FIG. 41.   FIG. 42.   FIG. 43.

FIG. 40.—Dilaceration of maxillary first incisor.
FIG. 41.—Dilaceration of another maxillary first incisor.
FIG. 42.—Dilaceration of maxillary first premolar.
FIG. 43.—Dilaceration of maxillary second premolar.

Etiology.—It is generally believed that a severe blow in the mouth may at times produce such an injury to the developing tooth or enamel and dentine germs as to cause—if the traumatism is not too great—that error of development to which the term dilaceration has been and is still applied. The incautious removal of the deciduous predecessor of a permanent tooth may likewise occasion it. The anterior teeth, with the exception, perhaps, of the canine, are most commonly affected.

Macroscopical Appearances.—The tooth may be curved at its neck or anywhere along the extent of its root. This bending may

be a mere continuous flexion of its outline, the apex of the root terminating in a normal manner; or there may be a double curvature. More rarely the crown appears to be joined to the neck of the tooth almost at a right angle or acute angle. Again, the crown may be apparently impacted in the body or root of the tooth. Superficially examined, there are no indications of morbid changes, except perhaps the occasional appearance of a mal-formed crown and a hyperplasic condition of the cementum.

Secondary Changes.—None.
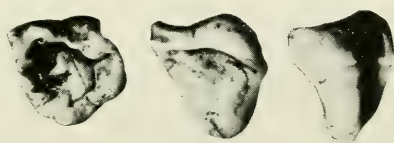
FIG. 44.   FIG. 45.   FIG. 46.

FIG. 44.—Dilaceration of a mandibular third molar. Occlusal surface showing convolutions of the crown with pigmented intervening fissures.
FIG. 45.—The same. Lingual aspect.
FIG. 46.—The same. Buccal aspect.

HISTOLOGY

At the seat of injury the dentinal tubules pursue a course which is far longer than normal. They are also strongly curved. The dentine at the point of curvature may sometimes exhibit signs of absorption on its cortical aspect, with subsequent deposition of cementum. The pulp canal retains its central position. The granular layer of Tomes is highly pronounced and thickened; and cemental hyperplasia, a result of chronic inflammation of the periodontal membrane, is present (see Fig. 48). The cementum of the parts unaffected by traumatism is normal and devoid of lacunæ, though its lamination is marked.

In dilaceration of the cervical region, a condition less common than that just described, the amelo-dentinal junction is bent on itself, and at times exhibits signs of absorption. This may contain hyperplasic cementum or enamel. The latter tissue may or may not be normal in structure. At times, however, a fissure or fissures, or even a canal, may exist, as in Fig. 39.

(ii) *Gemination*

Definition.—The union of two or more teeth. There are two kinds of gemination (i) True, (ii) False. Of these "True" gemination

is the term applied to those teeth which are joined to one another by
some cause which operates during developmental periods, and
without the aid of inflammatory conditions of the root membrane,
"False" gemination when the osteoblasts of the root membrane,
as a result of productive periostitis, have laid down an overgrowth
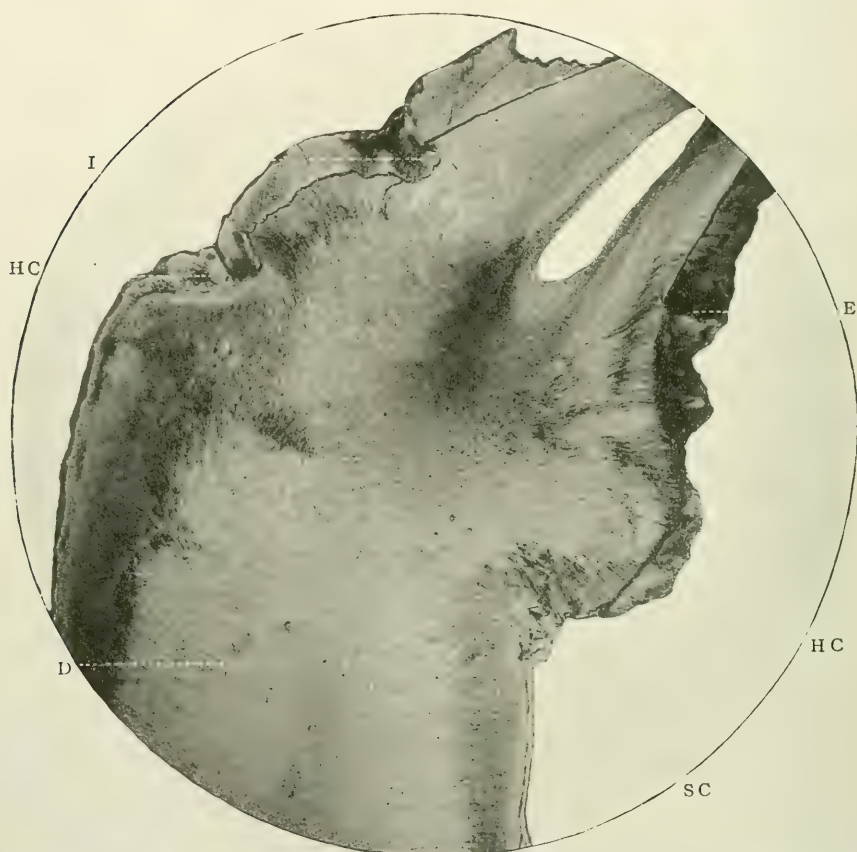
Fig. 47.—Dilaceration of the cervical region of a human incisor. Prepared
by grinding. Unstained. Magnified 12 times. e. Normal enamel; i. Involu-
tion of enamel into the dentine by penetration of the amelo-dentinal junction,
probably the result of absorption; h.c. Hyperplasic cementum at seat of dilac-
eration; s.c. Normal structureless cementum; d. Dentine.

of the cementum. It follows, from this definition, therefore, that in
the former, enamel and dentine may generally constitute the ma-
terial which unites the teeth, while in the latter hyperplasic ce-
mentum is the bond of union. As a rule members of the same
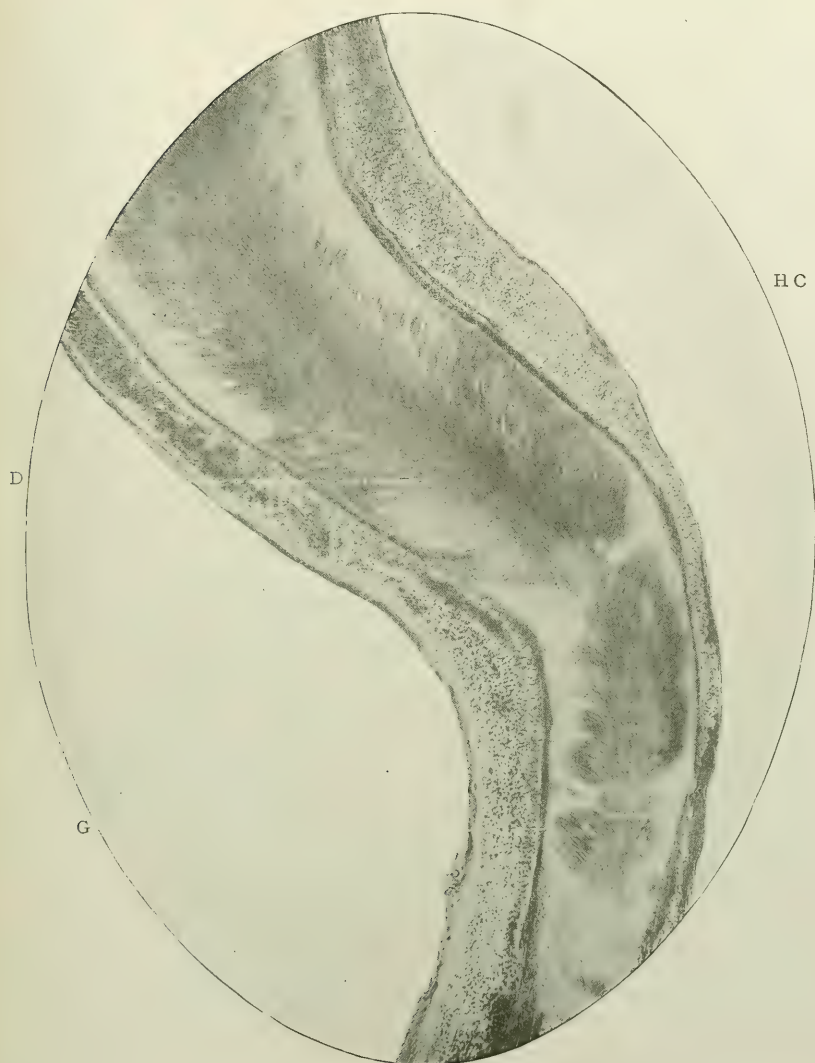
Fig. 48.—Dilaceration of the radicular region of a human canine. A second smaller curvature, not shown in the photomicrograph, existed near the apex of the tooth. Prepared by grinding. Unstained. Magnified 15 times. Shows enormous thickening of the hyperplasic cementum. D. Dentine; H.C. Cementum; G. Granular layer of Tomes.

dentition are affected, but it occasionally happens, as will be presently described, that individual teeth of the two dentitions are concerned. Thus a subvariety of "False" gemination may be known as "Diphyodontic" gemination.

Etiology.—The cause of the first variety is a little obscure. It may be due to dichotomy of the tooth germ at an early period of evo-

Fig. 49.—True gemination of permanent mandibular incisors. The labial aspect of the crown was excavated to ascertain the existence of one common pulp cavity.

lution, or it may be due to fusion of two or three tooth germs. This commonly occurs in a parallel direction; but not always. The teeth may be joined throughout their lengths, wholly or partially.

In the deciduous series, the mandibular incisors and canines are most frequently geminated. Thus the first and second incisors (Fig. 52), or the second incisor and canine, may be united, or more
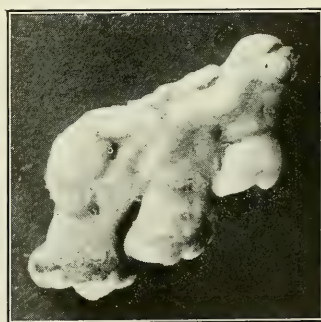
Fig. 50.—False gemination of three left maxillary molars.

rarely the two incisors and a supernumerary tooth (Figs. 54, 55 and 56). In the permanent dentition supernumerary teeth are often geminated to molars or incisors. Fig. 53 shows an example of a dwarfed supernumerary tooth geminated to a small third molar. It is a genuine case of true gemination, as the dentine is normal and presents no signs of the changes observed in certain odontomes.

Fig. 50 shows false gemination of three maxillary molars—a

somewhat rare condition. False gemination is due, as already indicated, to an overdevelopment of hyperplasic cementum—a result of productive periostitis of contiguous teeth. The bony septa have become absorbed and a solid union of the parts results.

FIG. 51.—False gemination of maxillary left second and third molars.

Geminated teeth occasionally occur in ovarian teratomatous cysts.

Macroscopical Appearances.—The external surfaces are irregular and exhibit considerable thickening over the region of the roots,
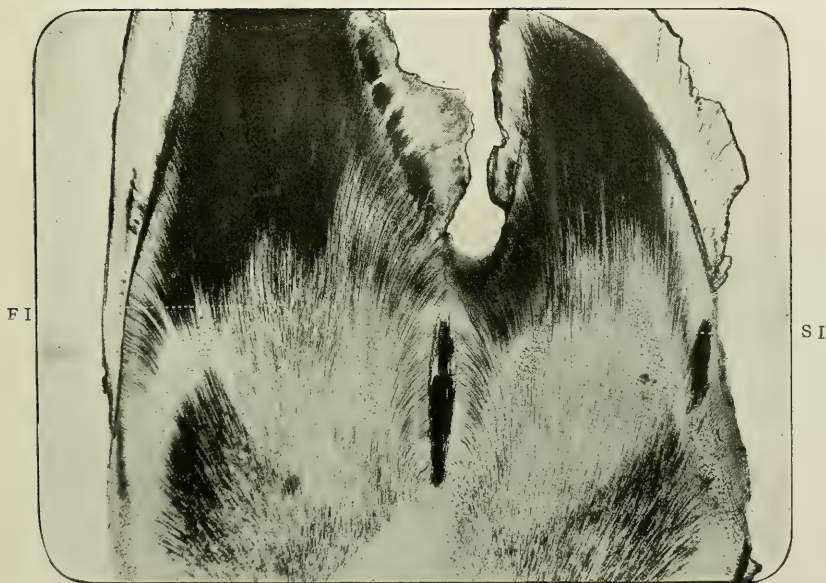
FIG. 52.—True gemination of deciduous mandibular first and second incisors. Coronal section of the teeth. Prepared by grinding. Unstained. Magnified 15 times. F.I. First incisor; S.I. Second incisor.

in the case of "False" gemination; a normal contour, with no appreciable addition in size, obtains in "True" gemination.

Secondary Changes.—Geminated teeth often show early symptoms of caries.
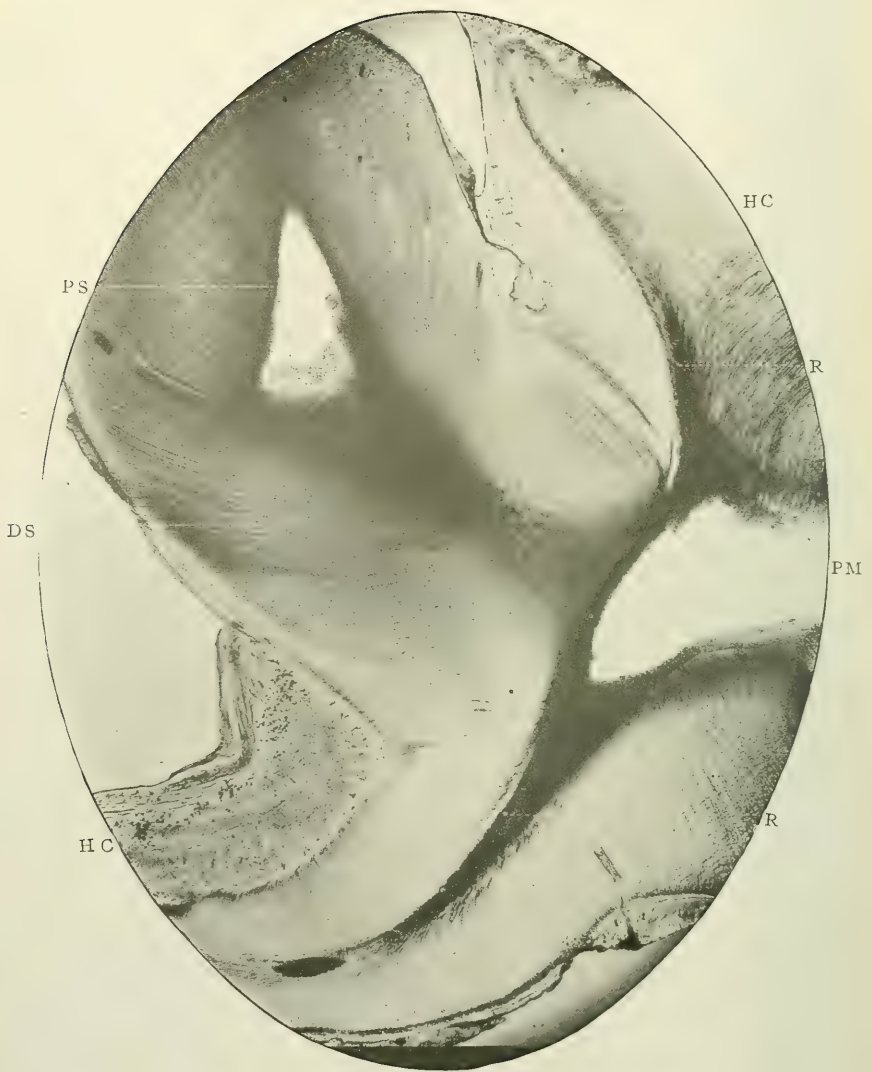
FIG. 53.—True gemination of a third maxillary human molar with an inverted impacted conical supernumerary tooth, as described in the text. Prepared by grinding. Unstained. Magnified 15 times. R. Pulp canals in the extremely divergent roots of the molar; P.M. Its pulp cavity; D.S. Dentine of supernumerary tooth; P.S. Its pulp cavity; H.C. Hyperplasic cementum.

HISTOLOGY

The diagrams reproduced in Figs. 54, 55, and 56 represent horizontal sections of true gemination of the mandibular deciduous first and second incisors, and a small supernumerary tooth, and reveal the facts that near the cervical margins three pulp chambers, separate and distinct from each other, each having normal radiating fine-tubed dentine exist. The enamel, as a narrow cervical ring, surrounds each segment of the tissue. Lower down two pulp chambers, one rounded and the other dumb-bell shaped, are formed (Fig. 55). The dentine, normal in every particular, intervenes between the pulp cavities. A section near the apex of the root shows that the two pulp cavities have become merged into one. In every instance the dentine and cementum are unaffected by morbid
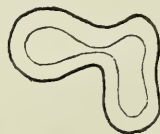
FIG. 54.   FIG. 55.   FIG. 56.

FIG. 54.—Horizontal section of three geminated teeth at cervical region, where there are three pulp chambers.
FIG. 55.—The same, showing two root canals.
FIG. 56.—The same, showing one root canal.

changes, the latter being merely a very attenuated annular layer of solid osseous material.

In Fig. 53 there is a strong suggestion of the impaction of a supernumerary tooth in the midst of the third molar producing a cleavage of the tooth germ. It is a curious anomalous condition, and probably unique.

The dentine exhibits the usual characteristics, but the enamel has been removed in process of grinding the specimen, while the cementum is hyperplasic, indicating, no doubt, that an injury had been received by the immature tooth germ. Embracing the neck of the supernumerary tooth the cementum of the molar is continued outwards, on one side as a fine triangular peninsula of hard tissue, on the other as a rounded cone-like elevation.

The root canals of the molar can be clearly followed almost to the apices of these cemental and dentinal projections.

4

Diphyodontic Gemination

There have recently come into the possession of the author two examples of this condition, in which, as already stated, a deciduous tooth may become firmly united to a member of the permanent series. The first, [which has no available history], illustrated in Figs. 57 and 58, is a left maxillary first incisor removed from the
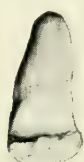
FIG. 57.   FIG. 58.   FIG. 59.

FIG. 57.—Diphyodontic gemination of a maxillary left permanent first incisor. Labial aspect.

FIG. 58.—Diphyodontic gemination. Same specimen as preceding. Mesial aspect.

FIG. 59.—Mandibular left permanent second incisor affected by diphyodontic gemination. Labial aspect.

mouth of a child of ten or eleven years old. The labial surface measures 18 mm. in length, it is 9 mm. wide, the normal measurements of a similar tooth being 12.5 mm. and 9 mm. respectively. The mesial surface of the root is 7 mm. the distal 6 mm. in extent. The root was deflected toward the mid-line, its apex was still patent. The

FIG. 60.   FIG. 61.   FIG. 62.   FIG. 63.

FIG. 60.—Similar to preceding. Lingual aspect.
FIG. 61.—Similar to preceding. Distal surface.
FIG. 62.—Similar to preceding. Mesial surface.
FIG. 63.—Similar to preceding. Occlusal surface.

length of the deciduous attached crown measured 10 mm., its width 6 mm.

The second case for which the author is indebted to Dr. Edward C. Kirk, occurred in the mouth of a girl aged fourteen years. It is a left mandibular second incisor. It was said to have erupted at the eighth year, and, so *the patient affirms*, followed a *deciduous prede-*

cessor. Its position and shape were annoying, as the incisive edge was directed toward the lower lip. On examination, the tooth was firmly socketted, although it was denuded of soft tissue on its labial aspect. The bone in this situation had been absorbed completely, and the gingival tissue was inflamed. The periodontal attachment on the mesial, distal and lingual sides was normal. The other teeth were normal, and there was no purulent effusion around them (see Figs. 59, 60, 61, 62 and 63).

The general appearances of these geminated teeth are shown in the photographs. The length of the whole tooth was 16 mm., its greatest width 3 mm. The width of the attached deciduous crown measured 6 mm. The length of the root itself was 10 mm. and it was divided longitudinally by an extensive fissure on both aspects. The crown of the deciduous tooth was placed at right angles to that of the permanent tooth, its concave lingual surface directed upward and its labial convex surface downwards.

(iii) *Lacunar and other Defects*

Definition.—Irregular spaces—not of an interglobular nature—which admit of no classification, found in the substance of the dentine.

Etiology.—Congenital defective nutrition or perverted metabolism of the pulp cells.

HISTOLOGY

Dentz, of Utrecht, has observed in the early-erupted teeth of a newly-born child certain curious defects, which are undoubtedly congenital in origin. Bodies containing apparently six or eight nucleated cells, which were closely applied to each other and somewhat resembling Pacinian corpuscles were discovered in the dentine, which measured about 0.25 mm. near the amelo-dentinal junction. The spaces were of great size (125μ in length), oval or bilobed in shape and outline (Fig. 64). A spiral structure, which might be in continuity with the dentinal fibrils could, with difficulty, be made out. It is possible that these defects marked a stage in the formation of interglobular spaces.

Ovarian teeth sometimes show areas of dentine where the tissue has undergone considerable metamorphosis. In Fig. 65 the tubes, which are very much smaller than those of the rest of the dentine are running centripetally, not centrifugally, from a point centred at
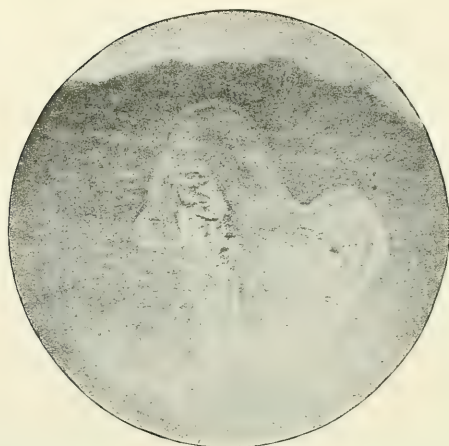
FIG. 64.—Lacunar defects in dentine. (*Dentz's specimen. Photomicrograph by Howard Mummery.*)
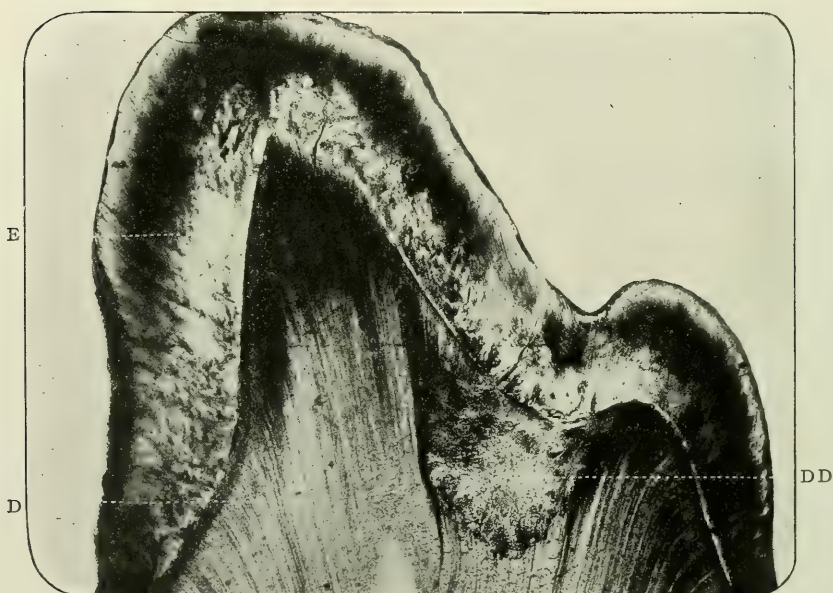
FIG. 65.—Coronal section of a tooth removed from a teratomatous ovarian cyst. Prepared by grinding. Unstained. Magnified 15 times. E. Pigmented enamel of defective quality; D. Dentine; D.D. Developmental defects in dentine.

the amelo-dentinal junction, below the sulcus of the crown[1]which intervenes between the two deformed cusps. In colour the abnormal patch resembles a pale brown, similar to that of the enamel. The foundations of the irregularity seem to be matrix of a poor quality, myriads of tiny interglobular spaces apparently covering it through all its extent. It bears some resemblance in outline to a sheaf of wheat.

The normal dentinal tubes are interrupted in their courses, which, however, are resumed in the immediate neighbourhood of the enamel.
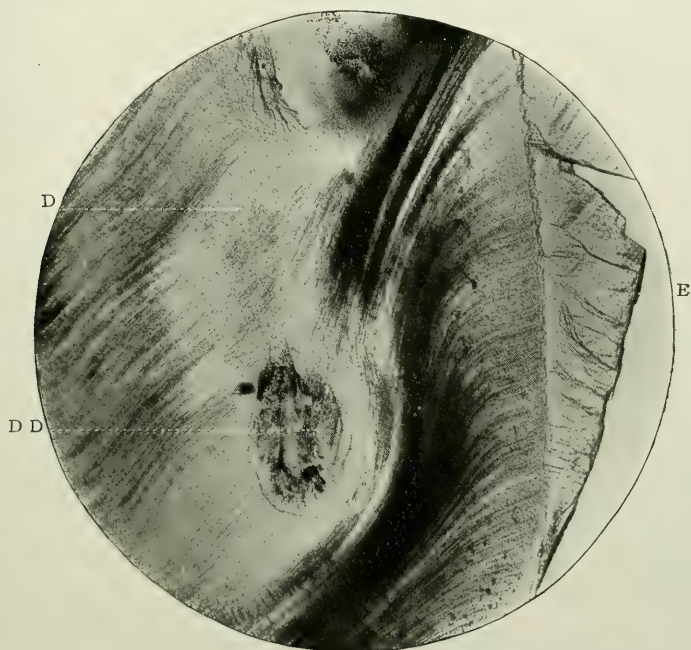
FIG. 66.—Structural defect in human dentine. Prepared as in the preceding figure. Magnified 40 times. E. Enamel; D. Normal dentine; D.D. Developmental defect.

The periphery of this interesting structure displays signs of lacunar absorption of the healthy tissue, while two or three large spaces, filled with amorphous material, occupy the position of the amelo-dentinal junction. The enamel itself is slightly defective in structure.

(iv) Congenital Pigmentation

Definition.—Deep brown coloration of the dentinal matrix and contiguous cementum of sound (*i.e.*, non-carious) teeth.

Etiology.—Some obscure congenital defect.

Macroscopical Appearances.—When a congenitally pigmented tooth is cracked in the jaws of a strong vice, its deeply pigmented characters are seen. The staining is uniform, not as in Pathological pigmentation, as described on p. 76, confined to one part entirely, or more pronounced in any particular locality.

The microscopical examination reveals nothing except pigmentation of the matrix.

(v) *Nanoid or Dwarfed Dentine*

Definition.—Aplasia or agenesia of the dentine of unerupted teeth, especially of their roots. It is not a simple atrophy of the parts. Atrophy implies a retrogressive metamorphosis occurring in tissues or organs which were originally well formed or "in pro-

Fig. 67.

Fig. 68.

Fig. 67.—Maxillary left first permanent incisor showing nanoid dentine. Labial aspect.

Fig. 68.—Same as the preceding. Lingual aspect.

portion to the age of the organism well grown."[1] Such a variation leads to diminution in size of the organ or structure. While the constituent parts shrink variously, through decrease in number and size, there is no marked alteration in the minute anatomy or in chemical composition.

Etiology.—An actual immature condition or imperfect development of the roots of teeth may be due to the fact that for some unknown reason, teeth have been made to assume abnormal positions in their crypts in the alveolar process of the jaws—positions in which eruption is impossible. The growth of the roots not being required to aid and complete this physiological process may suddenly cease. Failure of eruption is caused by failure of root formation, or perhaps *per contra*. The condition occurs in the young—about fifteen years. The maxillary incisors may be affected, in which case, if the alveolar process in the neighbourhood of the *columna naris* is dense and prominent, nanoid teeth may be suspected. A skiagram will reveal the condition.

[1] Hektoen & Riesman, "A Text-book of Pathology," Vol. i, 1901.

Macroscopical Appearances.—In the case figured, the actual measurements of the crown were 13 mm. in length. The root was 3.2 mm. on its labial, 6.1 mm. on its lingual aspect. The apical foramen was closed and the pulp canal shortened by about one-third of the length of the whole tooth. The proper length of the root should have been 20 mm. or more. The microscopical structure of nanoid dentine calls for no remark.

(vi) *Vascular Canals*

Definition.—Large, uniformly sized channels in the tissue passing from cementum to pulp, and containing, *post mortem*, a thrombosed blood-vessel.

Etiology.—The persistence at the periphery of the pulp of a large vessel or bundle of capillaries would probably give rise to this abnormality.

Vascular canals are occasionally associated with dilaceration of the teeth.

HISTOLOGY

Running from the pulp to the periphery, and piercing both dentine and cementum, the channels pass, sometimes sending out a branch or tributary which may end in a *cul-de-sac*. But the termination of the main canal itself is on the inner surface of the periodontal membrane.

A small artery with its attendant vein may be found *in situ*, the former empty of its contents, the latter sometimes blocked by means of a thrombus.

The condition is unconnected with cemental hyperplasia, or any of those pathological changes which sometimes occur round and about the pulp canals.

The presence of vascular canals in the hard tissues of the roots of teeth constitutes a grave source of danger if some devitalising agents happen to be used in connection with diseases of the pulps of teeth so affected. The condition cannot be diagnosed till after the removal of the tooth.

B.—ACQUIRED DISEASES

(vii) *Absorption*

This common pathological condition may take place from without or from within, *i.e.*, from the cemental or the pulp side.

Definition.—The removal of dentine, as part of a pathological process—in contradistinction to the physiological absorption of roots of deciduous teeth—not due to caries.

Etiology.—Chronic inflammation of the pulp or periodontal membrane from whatsoever cause may induce certain changes in the cells of the soft tissues, which, unduly stimulated, remove the hard parts in their immediate proximity. The absorption is probably due (i) to the activities of multi-nucleated giant cells (osteoclasts) as physiological factors, because these bodies, although
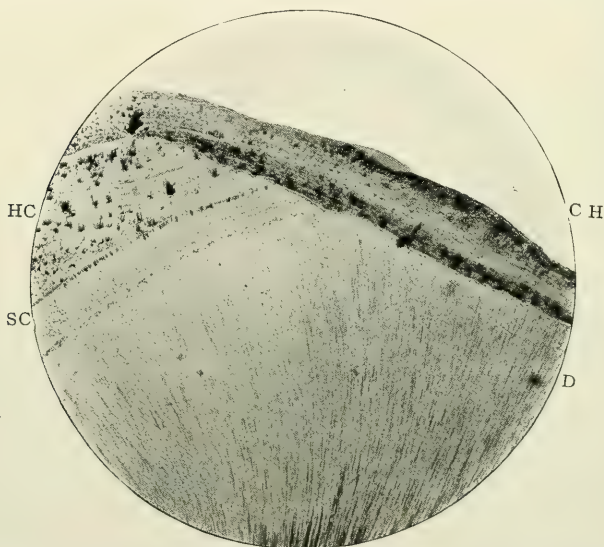
FIG. 69.—External absorption of dentine. Shows D. Dentine; S.C. Normal structureless cementum; H.C. Hyperplasic cementum. (*From a section in the collection of A. W. W. Baker.*)

they have never been observed in the pulp tissue, nor do they exist in the inner aspects of the periodontal membrane, yet are common enough in the bay-like recesses of the alveolar bone. The absorption may also, most probably, be correctly ascribed (ii) to the presence of absorption-leucocytes or phagocytes brought into physiological contact with the hard parts by means of the small cell infiltration of the soft tissues, the result of the inflammation.

Acute inflammation of the periodontal membrane accompanied by suppuration, as a result of impaction of a contiguous tooth may also induce absorption, probably (iii) by the action of acid pus merely dissolving away the lime salts from the organic matrix

near the seat of lesion, a pathological action resembling that which occurs sometimes in absorption of bone viz., halisteresis.

Pathologists agree that in the case of bone, pathological absorption is produced, either by (a) the agency of osteoclasts ("lacunar absorption"); (b) decalcification ("halisteresis"); or (c) granulation tissue, through the medium of certain round cells which possess a phagocytic function. It is exceedingly likely that these, or similar agencies, can produce like results with regard to the absorption of cementum and dentine.

Macroscopical Appearances.—When the apex or side of the root is affected, a roughening of the surface results. Thus "needle-point" absorption is common on the apex of the root.

No pigmentation occurs, and no caries.

Secondary Changes.—If the inflammation undergoes resolution, which it most generally does, organisation supervenes on an attack of chronic inflammation, and the cementum, more or less lacunated and laminated on the one hand, or a cementum-like substance, which has been termed "osteodentine"—a bad appellation—or even bone, well-organised and unmistakable—on the other hand, may be produced.

HISTOLOGY

(α) *External Absorption*

In the belief that osteoclasts properly so constituted do not exist in the pulp or the innermost zone of the root membrane, it is not surprising to find absence of Howship's foveolæ. It is true that more or less festooned outlines are met with in places, but they are nothing like those found in the physiological absorption organ of deciduous teeth or on the surface of the shafts of bone.

In this way, therefore, in the case of external absorption, a portion of the external surface of the dentine loses its normal outline, a break occurs in its continuity, the breach of surface being filled with irregularly-developed cementum. It may be hollowed out sometimes to any depth, or it may be absorbed in a straight line, as in a specimen of Baker's, of Dublin (Fig. 69).

Specimens of the first condition show the following details: The normal cementum and granular layer have disappeared; no sharp line of demarcation intervenes between the normal and the diseased parts; the dentinal tubes are cut off squarely with their course, and —a point of some interest—cemental lacunæ, large and confluent,

and not possessing prominent canaliculi are placed, in no great numbers, immediately on the cut ends of the tubes. Many lacunæ are of the abrachiate variety.

Both kinds of absorption may be illustrated by Dr. A. W. W. Baker's example. He describes it by saying[1] that "two-thirds of the circumference of the tooth show evidence of inflammatory action of a more or less chronic nature, that is to say, there can be traced zones of the cementum where the root membrane was in a healthy condition and deposited normal cementum, then periods where the root membrane became actively inflamed and absorption took place. . . . . The remaining third of the circumference of the root showed absorption by abscess of a severe and extensive nature, as not only the cementum but also the dentine was involved and absorbed. . . . . The distinct band of new tissue is here very well marked, and at first sight seems as if the section were folded upon itself" (see Fig. 69).

The writer considers that in this case osteoclasts are the predominating factors of the absorption. Sir John Bland-Sutton, in his "Introduction to General Pathology," p. 124, 1887, says: "In the giant cells (formed by the fusion of leucocytes, which have migrated from the new capillary blood-vessels of the granulation tissue of inflammation) we have the counterpart of the fusion of phagocytes; the large multinuclear osteoclasts seen in places where vertebrate bones and teeth are undergoing absorption, must also be placed in the same category."

If this interpretation is correct (and modern research would seem to confirm it), it is not difficult, it must be confessed, to comprehend not only how the new formation of osteoblasts in the membranes bordering the periphery of the cementum arises, but also in what manner their transformation into osteoclasts, and ultimately lacunar corpuscles, is brought about.

G. V. Black (*op. cit.*), speaking of absorption, states his opinion that the physiological absorption of deciduous teeth is precisely the same plan by which the roots of permanent teeth are occasionally, pathologically absorbed, either in part or completely. Nevertheless, though this writer evidently believes that cementum and dentine are both absorbed by means of osteoclasts, nowhere does he make a definite statement to that effect, nor do his figures show them. Indeed, while in every case his drawings of absorbed den-

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[1] "Advanced and Retarded Dentition." *Journal British Dental Association*, p. 432, 1902.

tine and cementum exhibit outlines somewhat simulating those of the foveolæ of Howship, he is careful to avoid the pictorial expression of an osteoclast—or, as some would call it, a cementoclast—filling up the excavations. One illustration exhibits particularly well these giant cells on the alveolar side of the root membrane, but they are omitted on the other.

Unique Extensive Absorption

A singularly instructive case of the absorption of the roots of many of the permanent teeth in one mouth which came under the personal notice of the author may be quoted. The case is possibly unique; and, possessing very extraordinary characteristics, its ætiology is so difficult to determine, and the actual pathological phenomena which it exhibits so full of significance, that, as a result, it is raised to a high level of interest and importance.

A man aged 44, has been under observation for a number of years, his mouth presenting very varied aspects as an infection advanced. *Family History:* The mother is edentulous: the father practically so. A sister, aged 25, has the right maxillary canine still unerupted, the cusp just appearing through the gum. There is no purulent effusion round her teeth. *Past History:* For fourteen years the patient, then being aged about 29-30, had a swelling under the mandibular first molar, which was very carious. Extraction under an anæsthetic failed to remove the tooth entirely. On examining the mouth at the time of operation, it was discovered that all the other molars—the upper and lower third molars not included—had their crowns only slightly erupting through the gum, although the premolars and incisors were fully developed and occluded in a normal manner. As the conditions were extremely uncommon, and as the swelling "did not go down," the patient was sent to the consulting surgeon of a Hospital, who, anxious to await further developments and give Nature an opportunity of correcting the abnormal state of the mouth and jaws, merely ordered an "ointment" for external use.

When the dental surgeon next saw the case an abscess had opened which would not heal. The patient was thereupon sent to the Hospital, and the surgeon for the day endeavoured to remove the roots of the tooth, but failed. He was then seen by the dental surgeon of another Hospital, and again operated upon, but with no greater success. Some long time afterwards the patient told his private practitioner that a piece of bone, or "something like it,"

came through the opening on the face, and then the abscess healed. All the partially-erupted molars except one have been shed spontaneously—*i.e.*, become loose, and been removed from the mouth by means of the fingers, and are in a more or less similar condition, showing signs of extensive absorption. The crown of a third right maxillary molar was, in December, 1908, felt with an explorer, buried in the jaw, the tooth itself erupting and spontaneously becoming shed by absorption of its roots, six months later.

The patient himself told the author that, with jaws closed, the fingers could be inserted between the occlusal surfaces of the molar teeth on both sides, showing that they never occluded in the normal manner, a space of one-sixteenth or one-thirty-second part of an inch intervening. For fifteen months there had been a certain amount of discharge of pus and swelling of the gingival tissues. Trismus was present at times to a slight degree, and the patient also suffered greatly from insomnia. The maxillary right canine was sound, but had an abscess associated with it, and was therefore somewhat loose. Having meanwhile been crowned, it is now quite firm. Practically there has been no pain throughout the whole course of the disease, the discharge of pus from the back of the mouth being the most prominent symptom. There has been no tartar to speak of, and the general condition of the oral mucous membrane is, and has been, fairly good. The patient had rheumatic fever when he was aged 33, is somewhat delicate, and probably neurasthenic.

At the time of examination it was noted that the mouth is clean and well cared for. Tartar is absent round the necks of the remaining teeth. Great absorption of the alveolar processes has occurred. Of all the maxillary teeth, the two canines, the second left premolar, and the root of the second right premolar remain, one of the former having been crowned. The latter, however, shows signs of chronic inflammation of the periodontal membrane especially on its distal aspect, probably through loss of bone-substance of the jaw. The mandibular alveolar ridges are flat; the teeth remaining *in situ* are the two second incisors, the canines, the two first premolars, and the left second premolar. The other teeth have been extracted from time to time, through loosening on account of the alveolar changes. Eruption of the right mandibular third molar is now taking place.

Macroscopical Appearances.—(i) *Left mandibular second molar:* On the occlusal surface of the crown there is a rounded cavity occupying the site of the postero-mesial cusp, its greatest diameter being

4.5 mm. and shortest 3.5 mm. Extending downwards and backwards and towards the lingual side, it avoids the pulp chamber and opens externally below the cervical margin on the posterior surface, by a small pin's head point, on the lingual surface by two sinuous perforations (Fig. 70), and in the radicular region in a large irregular shallow excavation, 9.5 mm. in length. The greatest area of absorption is found on the mesial aspect (Fig. 71). Here the enamel is
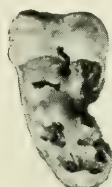
FIG. 70. FIG. 71.

FIG. 70.—Left mandibular second molar: lingual aspect.
FIG. 71.—The same tooth: mesial aspect.

unaffected, but immediately below, a large loss of dentine and cementum has taken place, measuring 12 mm. long by 8 mm. wide. The cementum is hyperplasic, the pulp cavity not exposed. The length of the tooth is 21 mm. There is no transparency of the roots, which are confluent through the hyperplasia of the cementum. One minute apical foramen can be detected.

FIG. 72. FIG. 73.

FIG. 72.—Right maxillary third molar: distal aspect.
FIG. 73.—The same tooth: buccal aspect.

(ii) *Right maxillary third molar:* The crown is flattened anteroposteriorly. A large amalgam filling, situated on the buccal surface, fills the site of the antero-external and antero-internal cusps. A discoloured patch of enamel is seen on the free edge. The mesial surface of the root is discoloured and presents three small hollows. A large area of absorption is observed on the distal and buccal surfaces (Figs. 72 and 73), the former being entirely excavated except at the extreme apical region. The roots are very hyperplasic

and reflected backwards. The apical foramina are closed and invisible. The whole of the palatine root has disappeared, the consumed surface measuring 12 mm. by 6 mm. by 5 mm. The length of the tooth is 21 mm.

(iii) *Right maxillary second molar:* The crown is roughly quadrilateral in shape. There is a gold filling on the morsal surface at the part between the antero-external cusp and the ridge joining the antero-internal and postero-external cusps. The crown is otherwise free from caries. The buccal roots are united together, rough, and greatly enlarged by hyperplasic cementum, the surface being very slightly attacked by the destructive process. The apical foramina are invisible. The palatine root is largely reduced in length by the absorption, slightly near the cervical region on the

FIG. 74. FIG. 75.

FIG. 74.—Right maxillary second molar: distal aspect.
FIG. 75.—The same tooth: lingual aspect.

lingual aspect, more so at the apical portion. The length of the tooth to the apices of the buccal roots is 18 mm., the palatine root measuring 16 mm. (Figs. 74 and 75).

(iv) *Left maxillary second molar:* The crown is non-carious. There is a tendency for it to become oblique in outline, its fissures and pits being pronounced. The enamel is entirely free from disease. The buccal roots are confluent, but discoloured. There are cavernous openings on all the surfaces, the greatest being over the outermost. It covers an area of 42 sq. mm. (Fig. 77). The pulp is not penetrated. The palatine root, exceedingly honeycombed, is reduced in length. There are several excrescences of hyperplasic cementum on the distal surface of the body of the tooth, at the junction of the roots (Fig. 76). The length of the tooth, on its buccal aspect, is 20.5 mm., and 17.5 mm. on lingual side.

(v) *Left maxillary third molar:* The crown here is non-carious, but flattened from before backwards. Enamel, while being discoloured, is intact, except undergoing a small amount of loss on the mesio-lingual side. Two small deep pits are observed on the buccal

aspect. All three roots are united and thickened. The buccal roots on the free surface exhibit an absorption area 7 mm. and 4 mm. across the widest and narrowest diameters respectively, while distally, an irregularly formed cavity is seen, and mesially, two small depressions, closely situated. The palatine root displays the ravages of the disease best of all. On the mesial aspect the dentine and pementum, covering a superficies of 13 mm. by 7.5 mm., have been
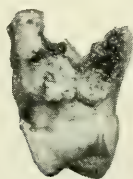
FIG. 76.   FIG. 77.
FIG. 76.—Left maxillary second molar: distal aspect.
FIG. 77.—The same tooth: buccal aspect.

removed (Fig. 79). The pulp is here apparently invaded. The length of the tooth averages 19.5 mm.

The edges of the excavations of all the teeth, on examination with a lens, are, generally speaking, rounded, everted, and smooth when dentine and cementum are destroyed; when enamel is involved, sharp and well defined. They nowhere exhibit the naked-eye ap-

FIG. 78.   FIG. 79.
FIG. 78.—Left maxillary third molar: mesio-lingual aspect.
FIG. 79.—The same tooth: mesial aspect.

cearances of having been produced by osteoclasts, as Howship's foveolæ are probably entirely wanting.

In reference to the present case several facts stand out strikingly. First, the molars erupted most imperfectly. Some unknown reason lies at the back of this. The ordinary physiological forces were not only retarded but in abeyance. They cannot have been affected by the deciduous dentition, as the permanent molars,

of course, come up behind the milk teeth. There must be some constitutional disturbance at work, acting in an extraordinary manner, in producing this grave defect in the assumption of their normal position in the dental arch of the masticatory organs. The history of the right maxillary third molar shows that its life duration only extended over about seven or eight months. There is complete absence of any of the usual causes of delayed or anomalous eruption of teeth, and its cause in the present case remains a mystery. Secondly, the molars were not devitalized—*i. e.*, "dead." In each, except one, the pulp was alive and protected by its wonderful physiological resistance from the attacks of the great pathological processes going on outside. Thirdly, the disease, whatever was its nature, was practically confined to the molar region. None of the other teeth were similarly affected. This is the most inexplicable part of the case. That the infection, if it was induced by pyogenic micro-organisms in the first place, should limit itself to the posterior parts of the oral cavity is truly surprising. Fourthly, the presence of pus in large quantities at times was a prominent sign and, no doubt, associated with the granulation tissue produced around the roots of the teeth. Fifthly, the molars, having shared a common affection which was spread over a considerable number of years, became shed spontaneously.

It is difficult, as has already been pointed out, to decide on the ætiology of this absorption. The author puts forward his opinion as follows: There were two factors probably which played an important predisposing part in the production of the disease—viz., (1) the disuse of the teeth through lack of occlusion, and (2) constitutional debility. The probable sequence of events would be, a bacterial invasion of the periodontal membranes of the functionless molars, setting up a chronic periostitis which resulted in organization and hyperplasia of the cementum. The bacterial influences not having been withdrawn, the chronic periostitis was further changed into granulation tissue which, occupying the sockets of the teeth, by means of small round cells possessing phagocytic properties, removed not only the alveolar bone but almost equally the dentine and cementum of the teeth.

(β) Internal Absorption

There are evidences in absorption from the pulp side that absorption-leucocytes (osteoclasts) perform the functions of removal

of the dentine, and traces of the effects of chronic inflammation of the pulp are not wanting. Internal absorption is accompanied by obliteration of the original outline of the root canal, the resulting cavity being irregular in contour and enlarged to a varying degree. The more or less semilunar excavations may be confined to one or more portions of it, and produce subsidiary canals which run at angles—acute, right, or even obtuse—to the original pulp chamber.

Caush[1] has well investigated this subject. He writes:—"These semilunar excavations vary much in size as well as in number,
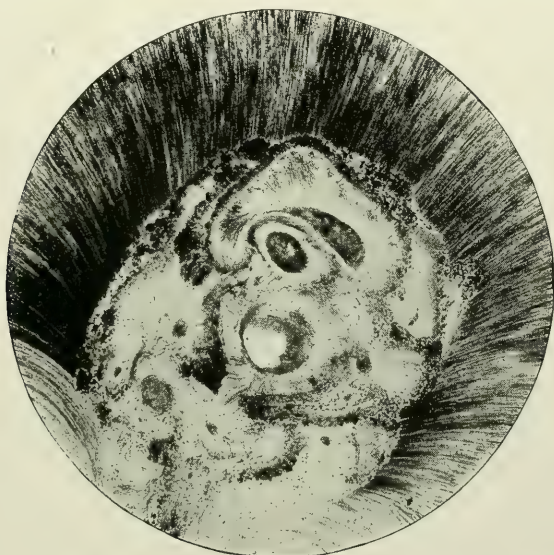
FIG. 80.—Internal absorption of dentine, with deposition of cementum-like material in the pulp cavity. Magnified 45 times. (*From a section in the collection of Douglas Caush.*)

varying from a single light dip or depression, as found in the earlier stage, to the numerous excavations producing the complex and irregular outline as seen in the advanced stage."

Very often this removal of tissue is followed by deposition of dense osseous material, a sharp line of distinction between the new and the old marking off this new development (Fig. 80). The adventitious structure is devoid of tubules—thus differing from many forms of adventitious dentine—and is, therefore, not dentine; but it is composed of lacunæ and canaliculi imbedded in a granular

[1] Trans. World's Columbian Dental Congress, p. 114, *et seq.*, 1894.

5

matrix, as in hyperplasic cementum. The lacunæ vary in number, size and position: thus they may be placed very closely together, or scattered throughout the mass. In the former they possess short, in the latter elongated canaliculi.

In explanation of the *modus operandi* by which these changes are wrought, Caush describes alterations in the shape of the odontoblasts, which, under the influence of hyperæmic or inflammatory conditions of the pulp undergo sub-division by mitosis. The cells in contact with the dentine begin to absorb that tissue, their function being more rapidly carried on, and far more reaching in its effects, if the dentine forming the boundary of the root canal happens to be somewhat poorly calcified or developmentally defective. Caush inclines to the belief that the odontoblasts can become converted into osteoclasts, which, later on (that is to say, when their absorptive energies are concluded), become again changed to formative cells and manufacture the osteoid cementum-like tissue. He, however, gives no drawing of giant cells. They are not required: the mere effect of the inflammatory products themselves, viz., the phagocytic leucocyte, being sufficient, *per se*, to produce the excavation.

In this connection, an instructive case, described by F. J. Bennett in *The Dental Record* for June, 1899, may be quoted, summing up, as it does, very succinctly the salient pathological features of internal absorption. He writes:—

"Whilst examining a collection of old teeth, the following specimen came under my notice, which is of interest, partly from its presenting a pathological condition of uncommon degree, and also as illustrating the steps employed by nature for the removal and repair of injured tissues.

"The tooth was a well-formed lower molar which had been attacked by caries upon the proximal surface at its junction with the cervical region. . . . . On section, the pulp canals were found to be empty, and showed no signs of having been treated. The surface of the roots presented the usual appearance of long continued chronic inflammation, being thickened with patches of porous lightcoloured cementum, and having vascular canals here and there penetrating the substance of the tooth. Indeed, it was due to one of these apertures of unusual size, and with rust coloured margins, that attention was first called to the case. Situated midway between the neck and the apex of the anterior root, it was found on section to terminate in two large cavities representing the pulp canal, which at

this point had become extended in various directions greatly beyond its original size.

"Under the microscope, it was found that the cavities and the channels leading to them were everywhere attacked by absorption, presenting the conspicuous lunated outlines known as Howship's foveolæ or lacunæ. The margins were also stained with the rust colour of hæmatin crystals.

"The cementum covering the root was thickened, and towards the apex there were signs of alternations of absorption and deposition.

"This direct and extensive attack upon the interior of the tooth suggests the existence of some source of irritation within the root injurious to the surrounding cementum and periosteum; and further, that it was for the removal of this source of mischief that the process of absorption was set in action.

"Quite recent investigations have shown that leucocytes are the essential agents in the process of absorption, the mass of highly vascular granulation tissue found lining these absorption cavities being the means by which the absorption cells are brought into actual contact with the affected tissue. These absorption leucocytes or phagocytes, migrating from the capillary vessels of the granulation tissue singly, or uniting together to form giant cells, attack and take into their interior the particles to be removed.

"The recognition of the nature of the osteoclasts as originating in the leucocytes may be considered as a great step in advance, as it leads one to regard many of the processes at work in the hard and soft tissues as essentially similar in nature. According to Ziegler, 'Recent researches into the absorption of bone appear to place it on a level with absorption of other tissues, and to view them all from the same standpoint.'[1]

"Although it is common in the soft parts and in bone to find diseased tissue becoming detached in the form of a slough, or of a sequestrum at its line of junction with the healthy tissue, by the interposition and activity of the phagocytes, this is possible only in tissues possessing a vascularity sufficient for the conveyance of the cells to this situation. The cementum, however, possesses no such system of capillary vessels throughout its substance for the circulation of blood cells, as in the case of the Haversian canals of bone; consequently this method of removal of diseased tissue by exfoliation does not occur. The process adopted in this instance is essen-

[1] Ziegler's "General Pathology and Anatomy," p. 160.

tially similar to that occurring in the molecular absorption, or true caries, of bone. The phagocytes channel a passage . . . . the channel becomes lined with the granulation tissue which penetrates the dentine and ultimately spreads itself over the surface of the root canal, allows the migrating cells to absorb and remove the diseased dentine down to the surface of the living cementum.

"I am inclined to regard this specimen as illustrating a conservative aim on the part of Nature. The form of the absorption is different from that usually seen in permanent teeth which are being shed. In the latter case, the root is either shortened or reduced in circumference, and the cementum is equally—if not more—absorbed than the dentine. In this specimen the struggle to maintain the cementum in an efficient condition is exemplified by the patches of removal and redeposition of cementum which occur in various places.

"I have not met with an instance in which repair has followed to such an extent that the root cavity comes to be again occupied by fresh nutrient tissue, but a remarkable specimen described and figured by Salter[1] inclines me to the belief that this may sometimes take place. In this case well-formed bone with Haversian systems had come to occupy a large portion of the pulp cavity, which latter bore traces of previous lacunar absorption. The pulp cavity had three circular apertures entering it from the external surface a little below the neck, and these were also lined by bony tissue. The ossification of granulation tissue in my own specimen would probably have led to similar appearances."

True bone, identical with the compact variety, may fill, under rare conditions, all the interspaces of internal absorption of dentine. In such cases, the Haversian systems are large, the lacunæ numerous, and the osseous lamellæ pronounced, while large irregular spaces probably contain in the recent state portions of the included pulp. Four well-authenticated specimens are on record, one described by Messrs. John Ackery and J. F. Colyer (*Trans. Odonto. Soc. of Great Britain*, p. 66, 1893), a second by Salter, a third by Tomes (*Trans. Odonto. Soc.* p. 178, 1899), and the last now published with a photomicrograph for the first time.

The first case was as follows:—

A partially-erupted premolar in the mouth of a patient of 33 years of age, exhibiting caries, was removed on account of irregu-

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[1] "Dental Surgery and Pathology," p. 79.

larity in position. The root was only two-thirds completed. It was difficult to say whether this diminution in size was due to arrested development or absorption; but the latter was the more plausible theory.

"The transverse sections showed that the dentine contained in its midst true bone. In one, taken near the crown, two or three small canals were seen containing tissue exactly like bone. In a lower section, a large portion of the dentine had been replaced by a tissue very irregular in character, but being, in places, of a distinctly osseous character. To the side of this, but yet separated from it, was another oval space containing distinct osseous tissue. In a third section these two spaces had fused together, and the first had progressed much further into the dentine. The tissue towards the pulp chamber still retained the irregular character already spoken of, but in the portion most distant from the pulp there was distinct osseous tissue with well-marked Haversian systems. The next section showed a still greater portion of the dentine replaced by true bone, the irregular tissues only being apparent in that part nearest to the pulp chamber."

Salter's specimen[1] was a molar having three canals piercing the cervical region just below the edge of enamel. Vertical sections were made, and revealed the following:—The substance around the canals was yellower and more translucent than the normal dentine, extended for some distance into the crown, and down the side of one of the roots. Examination with the microscope proved this to be cancellous bone in all its salient features—small, uniformly sized and regularly arranged lacunæ, with few small canaliculi, and even and regular lamellæ placed parallel with the outlines of the cancelli. The line of demarcation between bone and dentine was strikingly sharp, though here and there isolated patches of the latter were inextricably mixed up with the former.

For the last example, the author is indebted to the courtesy of Mr. H. R. Pring. A maxillary first incisor from an old patient became loose, and on removal was seen to have an absorbed root. Median coronal sections, viewed microscopically from within, gave the appearances of a spicule of bone in dentine. Histologically, the tissue was true compact bone, as is seen in the photomicrograph (Fig. 81). It requires no special description.

[1] *Op. cit.* p. 97.
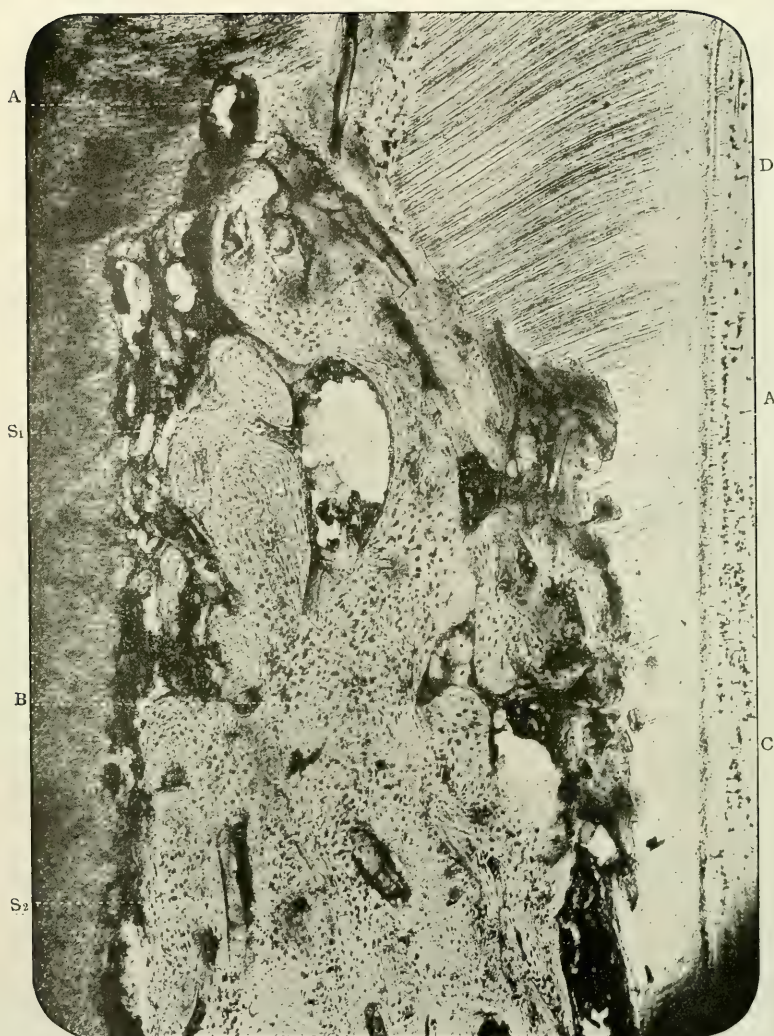
FIG. 81.—Vertical section of human incisor with internal absorption of dentine, and deposition of true compact bone. Prepared by grinding. Stained with impregnation by coloured collodion. Magnified 50 times. D. Primary dentine; A. Area of absorption; B. Compact bone; S_1. Large Haversian canal; S_2. Small Haversian canal; C. Hyperplasic cementum.

(viii) *Adventitious Dentines*

Definition.—Tissue of a pathological nature which has been added, in the course of dental caries, and in erosion, to the primary or first-formed dentine.

Etiology.—Inflammatory conditions of the pulp, the result of penetrating or deep caries of the dentine produces stimulation of
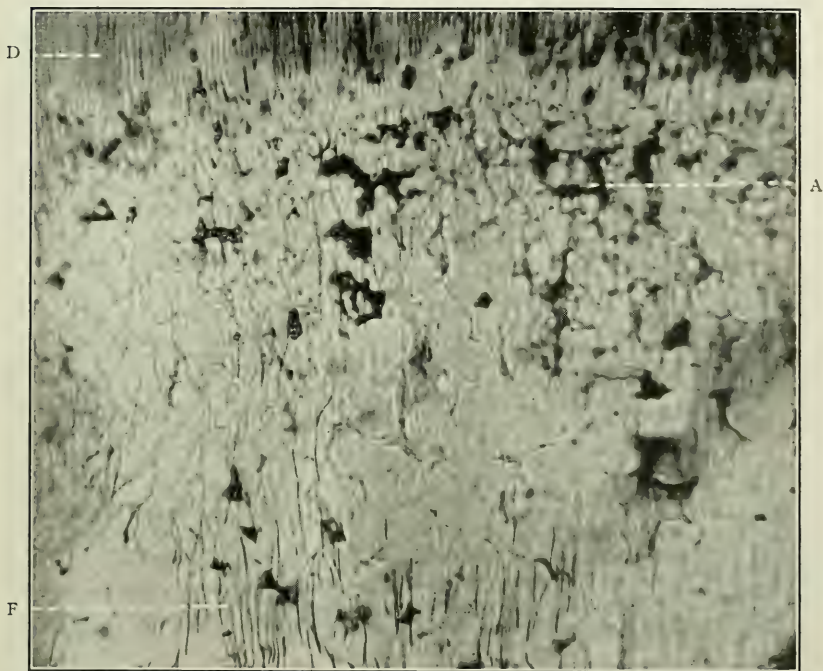
FIG. 82.—Areolar adventitious dentine. Magnified 500 times. D. Primary dentine; A. Areolar spaces filled with detritus; F. Connective-tissue fibres.

the peripheral cells—not odontoblasts—of the pulp, which then proceed to lay down a new form of dentine.

Macroscopical Appearances.—None.

Secondary Changes.—Certain varieties may, under the influence of advancing caries, become infected with micro-organisms. Of all the varieties, except the hyaline, to be presently noted, each is liable to this infection.

HISTOLOGY

Microscopically considered, there are five varieties:—(α) areolar, (β) cellular, (γ) fibrillar, (δ) hyaline, and (ϵ) laminar.

(α) *Areolar*

Its general characteristics resemble those of dentine filled with interglobular spaces (Fig. 82). It intervenes, as a tissue of repair, between a carious cavity and the pulp. Modified tubules may or may not accompany it. When they do, they are sparingly filled with round and rod-shaped micro-organisms, enlarge greatly as they extend inwards, and terminate with wide open mouths at their pulpar extremities. Probably this widening of the tubes is due to partial softening of the intertubular matrix.
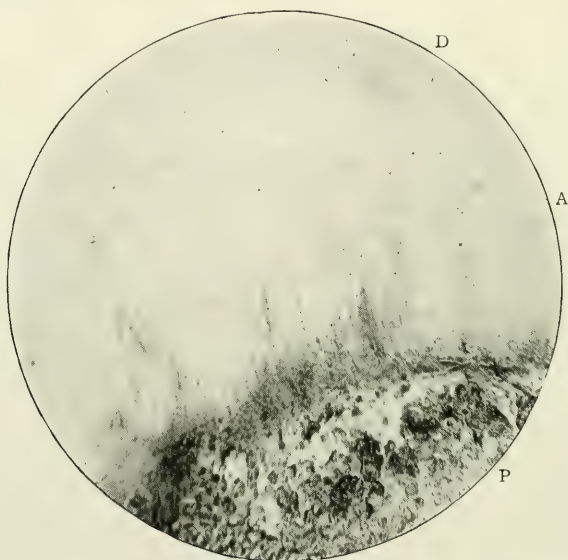
FIG. 83.—Areolar adventitious dentine. Magnified 250 times. D. Primary dentine; A. Areolar dentine; P. Pulp tissue.

It forms a thick sheet of hard material, devoid of ordinary dentinal tubules, being constructed of partially-fused calcospherites in the ordinary way. Its free edge is covered with round cells. Hyperæmia of the pulp accompanies its formation. It is the most commonly recurring of all kinds, and may be associated with the fibrillar variety, as in the foregoing illustration.

(β) *Cellular*

This is probably the same as that just described, with the addition of the presence in the matrix, of cells which may be fusiform or round.

The former are common when an areolar condition is present, the latter when a hyaline matrix invests certain round or spindle cells of the pulp. They are caught unawares, so to speak, in the osseous flood poured out round and about them.

The cells vary very much in size, the innermost being six or eight times as large as the smaller ones near the dentine. Towards the former side, rows of encapsuled cells may exist, some being multinucleated as if they had undergone mitosis. The surrounding pulp tissue, in immediate relationship with the new deposit, consists mainly of fusiform cells arranged in bundles interspersed with small
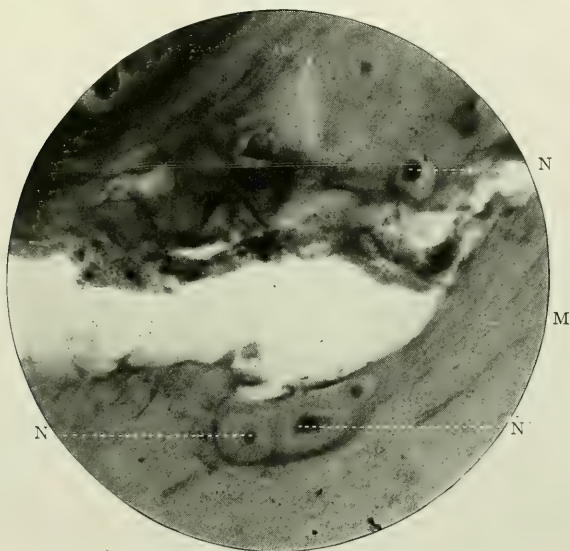
FIG. 84.—Cellular adventitious dentine. Magnified 250 times. N. Nuclei of encapsuled cells; M. Matrix.

round cells, the former possessing oval and the latter rounded nuclei.

(γ) Fibrillar

Fibrillar adventitious dentine bears a close resemblance to ordinary dentine; but the tube-like structures are much finer and less regular than in ortho-dentine. It is extremely probable, though not admitting of easy demonstration, that the lines are actually connective tissue fibres, and not hollow tubes.

The dentine is deposited in layers, and presents a characteristic

fibrillar structure. It is observed in cases of chronic caries. On the border line of the hard and soft parts, the connective tissue formation of the dentine is strikingly established.

(δ) *Hyaline*

This kind of new dentine has, as its favourite site, the base of a carious excavation into the pulp cavity. It may be irregularly rounded in shape. Its structure, in some places, conforms to that of a more or less homogeneous ground-glass-like matrix similar to
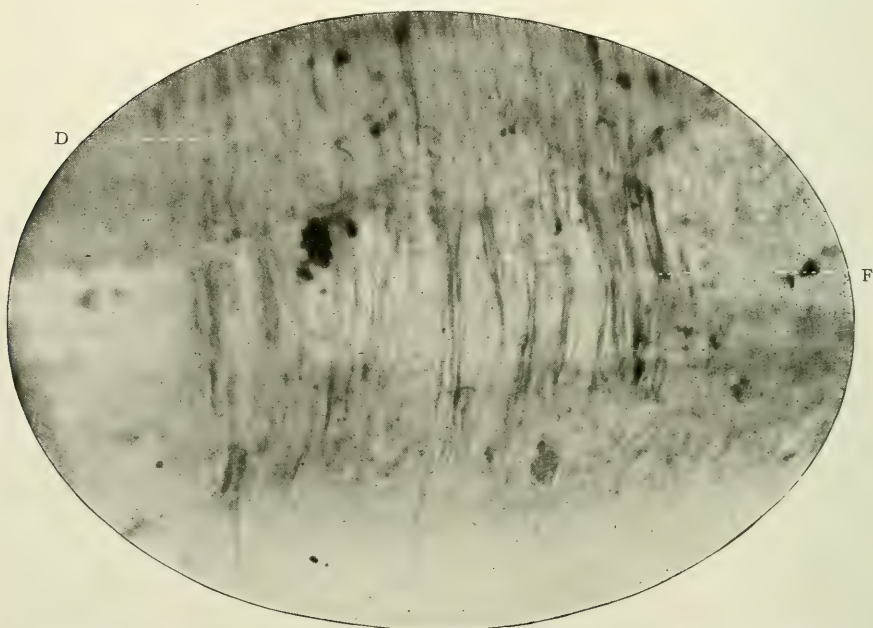
FIG. 85.—Fibrillar adventitious dentine. Magnified 250 times. D. Primary dentine; F. Fibrillar dentine.

that of hyaline cartilage; in others, it has a distinctly granular or fibrous appearance.

Distributed at its margins, cellular adventitious dentine may be often observed.

It is associated with chronic inflammation of the pulp, especially when this has fungated through the opened pulp chamber, granulated and produced a soft tumour in the carious cavity of the crown of the tooth—productive inflammation of the pulp, formerly called "polypus."
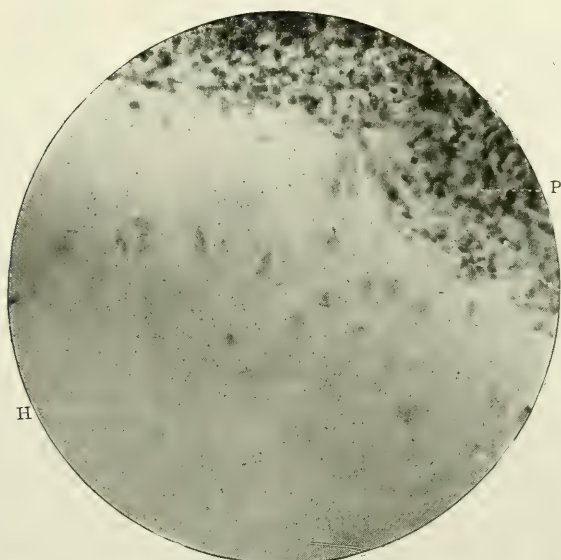
FIG. 86.—Hyaline adventitious dentine. Magnified 250 times. H. Hyaline dentine; P. Pulp tissue.
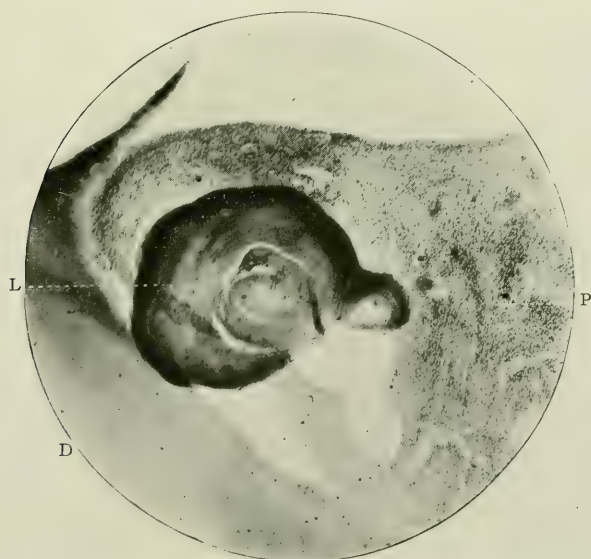
FIG. 87.—Laminar dentine. Magnified 250 times. D. Primary dentine; L. Laminar dentine; P. Pulp tissue.

(ε) *Laminar*

The variety thus described also may be associated with it in laminated form, in which patterns simulating pulp nodules appear. In fact, it is possible that these bodies are metamorphosed pulp nodules.

(ix) *Pathological Pigmentation*

Pigmentation of the dentine may be partial or complete, pink or green of varying shades or degrees. It never exists alone, the cementum and very rarely the enamel sharing its staining.

Definition.—A permanent coloured condition of the dentine.

Etiology.—Never a congenital defect, it differs from like conditions of enamel, in the fact that it may be produced in one of two ways, each of which leads to different results. Thus: If, as a consequence of a blow on, or damage to, the pulp through mechanical or chemical agencies, an artery is ruptured, there may be, under favourable circumstances, an escape of oxyhæmoglobin into the neighbouring structures. The colouring matter of the blood permeates the dentinal tubules, and sets up both superficial and deep areas of hæmatogenous pigmentation. Here the colour is pink.

In the second case, staining of a green character is due to the discoloration of the tissues, either through death of the organic parts, or through the introduction into the root canals of some filling material, as is induced by some kinds of amalgam, especially those, it is believed, which contain salts of copper.

Precisely how this is brought about is unknown.

Macroscopical appearances are unnoticed till the tooth has been removed from the mouth. The cementum is stained, and on breaking the dentine in a vice it also is found coloured throughout.

(x) *Senile Dentine*

While enamel, once fully formed, is incapable of undergoing any further changes incidental to old age, dentine, especially in the radicular regions of teeth, is often affected by them.

Definition.—The dentine of old age.

Etiology.—The cause may be attributed to a weakened or dimin-

ished nutritive supply, producing trophic changes primarily in the pulp and secondly in the hard tissues.

Macroscopical Appearances.—The dentine and cementum are rendered yellow or brown, and the apices of the roots become semi-transparent, or in long-continued cases quite transparent.

Secondary Changes.—None.

HISTOLOGY

The matrix is granular; the tubes are not narrowed, but are apparently absent. This is not, however, the case; an internal
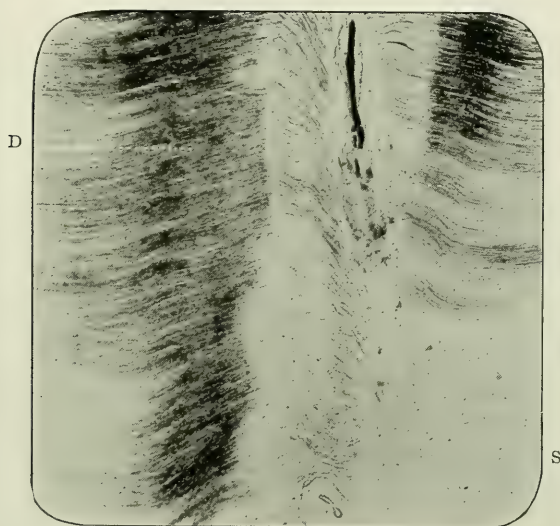
FIG. 88.—Senile dentine. Magnified 45 times. D. Primary dentine; s. Primary dentine which has undergone senile changes; P. Calcified pulp.

change has made them by transmitted light assume or approximate to the same refractive index as the matrix. They cannot be stained. Little is known with certainty as to what has occurred, but it is extremely likely that they have become occluded throughout their extent, wholly or partially, by a deposition of calcified material within their walls. They are no doubt solid, and incapable of transmitting sensations to the pulp.

The tubules and their contents were believed to undergo fatty degenerative changes by Heider and Wedl, who, in their well-
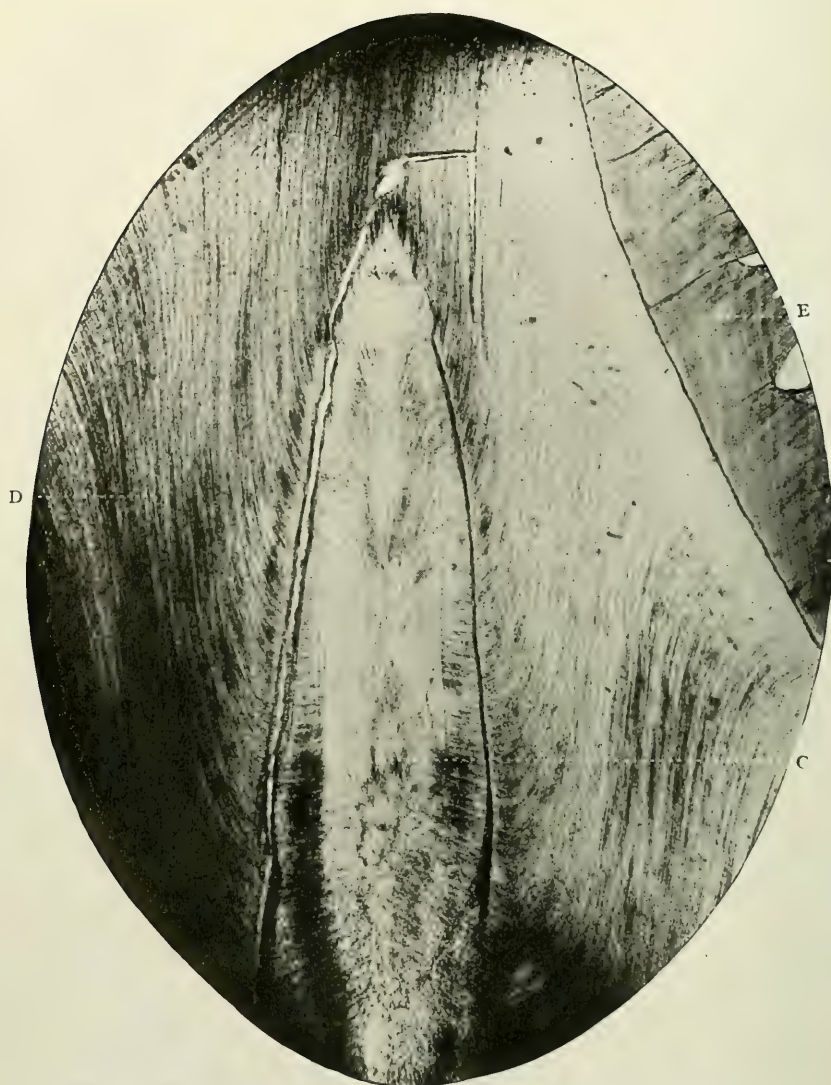
Fig. 89.—Vertical section from a senile tooth. Prepared by grinding. Un-
stained. Magnified 35 times. E. Enamel; D. Primary dentine; C. Calcified
pulp. (*From a specimen in the collection of G. W. Watson.*)

known Atlas, in Fig. 75, describe a varicose condition of the tubules. The enlargements contained fatty shining granules, arranged in *pater-noster*-like rows.   They were associated with free fatty drops.

Accompanying senile dentine, the pulp cavity is generally filled, as in Figs. 88 and 89, with a dense deposit of secondary dentine, and there is nearly always present a certain amount of cemental hyperplasia.

CHAPTER III

THE PATHOLOGICAL CONDITIONS OF THE CEMENTUM

Microscopical Elements in:—(i) Cemental nodules; (ii) Ankylosis; (iii) Hyperplasia; (iv) Senile cementum.

A.—DEVELOPMENTAL AFFECTIONS

(i) *Cemental Nodules*

Definition.—Sessile circumscribed tumours of the cementum, which, in man, are extremely rare. They may be called "cementomata."

Etiology.—They are produced by local development and calcification of the osteoblasts of the periodontal membrane.

Macroscopical appearances are the same as in enamel.

Secondary Changes.—None.

HISTOLOGY

The dentine is normal and the granular layer of Tomes presents no changes.

The cemental matrix is structureless, and extends as a thin layer over the dentine.

The neoplasm is made up of arcuate bands of matrix with laminæ and canaliculi. Fairly numerous, the first formed layers are dense and more lacunated than the youngest portion. The inflammation of the periodontal membrane has undergone resolution, indicated by the formation of the homogeneous layer of cementum most externally. The photomicrograph, Fig. 90, shows a nodule which existed at a locality 6 mm. below the cervical edge of a mandibular molar. The specimen is believed to be unique.

B.—ACQUIRED DISEASES

(ii) *Ankylosis or Synostosis*

Definition.—Solid osseous union between roots of teeth and their containing alveolar sockets. The condition must not be con-

fused with "False" gemination of teeth, for, in the latter, the pre-existing sockets have been completely destroyed by absorption processes.
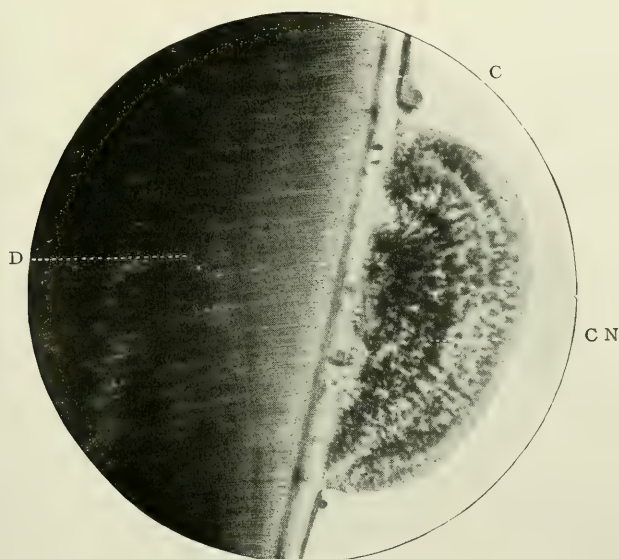
FIG. 90.—A cemental nodule. Prepared by grinding. Unstained. Magnified 15 times. D. Dentine; C. Normal cementum; C.N. Nodule. *Cf*, FIG. 10.

FIG. 91.—Mesial aspect of a human second maxillary molar with ankylosis of its root to the jaw. Slightly enlarged. (*From an original photograph.*)

Etiology.—Inflammation of the periodontal membrane aggravated or accelerated, or even perhaps originally produced by certain constitutional dyscrasia, such as gout, rheumatism or other trophic neuroses produces it. The probable course of the pathological phenomena is as follows:—The root membrane having been attacked

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by septic or infective micro-organisms, during the course of the inflammation, becomes injected with blood due to hyperæmia and rapidly becomes infiltrated with inflammatory products. Suppuration ensues. The whole of the membrane then undergoes conversion into a thick mass of granulation tissue. The adjacent bone of the jaw soon gets permeated with inflammatory products, and undergoes a kind of rarefying osteitis, or ulceration. Thus a small-cell exudation invades the Haversian canals of the alveolus, and together with osteoclasts which may be increased in number, destroys portions of the bone, and soon is changed into granulation tissue, which joins with the granulation tissue of the root membrane.

On the cementum side these pathological phenomena do not make such marked progress. But it is conceivable that the small-cell exudation excavates the periphery of the cementum, producing a roughening of the exposed surface, and that the granulation tissue of the membrane rapidly passes into and fills the irregular spaces so produced.

The layers of granulation tissue having presently united, a fibrous stage is in time reached, and then follows ossification of the entire parts.

Macroscopical Appearances.—Roots of teeth so affected present a bony, rough, dull growth on their extremities; they may be bathed in purulent effusion (Fig. 91).

Six cases of synostosis of teeth in man have been placed on record: one occurring in the practice of W. A. Whatford, a second by Storer Bennett, a third by M. Choquet, a fourth by G. W. Watson, a fifth and a sixth by the author. As possessing many points of interest, the latter three may be now described.

MR. WATSON'S CASE

The size of the mass of bone attached to the tooth was remarkable, and some idea of its bulk may be obtained from the following particulars:—Its greatest diameter extended in length 17 mm.; the greatest depth from apex of the posterior root equalled about 4 mm. The inter-radicular regions measuring from the distal ending of the dentinal tubes was about 5 mm. See Figs. 92 and 93.

HISTOLOGY

The specimens were made from a carious mandibular molar.
The pulp canals showed no signs of change.

The cementum at the neck of the tooth was normal, that is to say, structureless, no lacunæ being present. The lamellæ and incremental lines were, however, highly pronounced.

The root membrane was normal just below the cervical margin; at some considerable distance above the apex of the anterior root it is deflected for a short length over the bony alveolus.

The granular layer of Tomes is strikingly broad, and its constituents clearly defined.
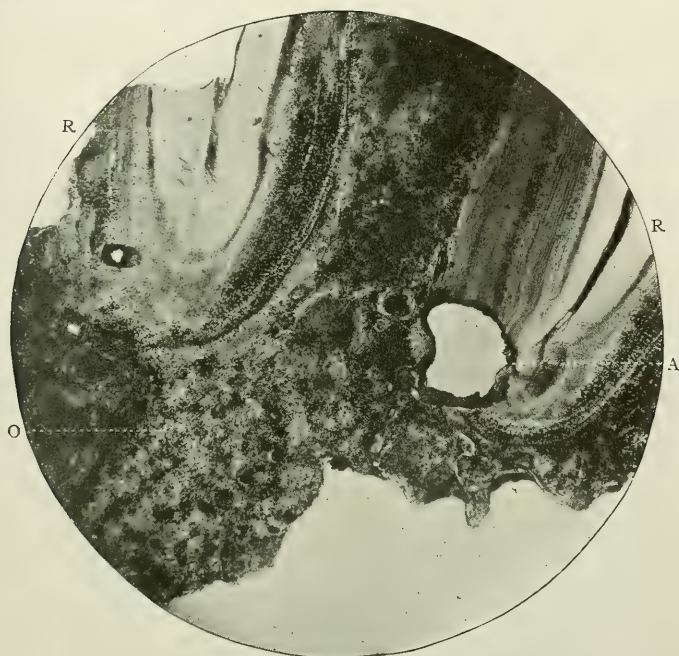
FIG. 92.—Ankylosis of the roots of a human mandibular molar. Prepared by grinding. Unstained. Magnified 12 times. R. Apices of the roots; O. Osseous tissue; A. Abscess cavity. (*From a specimen in the collection of G. W. Watson.*)

The cementum towards the apical space becomes trebled or quadrupled in width, with marked penetrating fibres, but the innermost zones are devoid of lacunæ and canaliculi. The middle and outer zones are characterised by the presence, arranged in parallel stripes, of incremental lines with dense striæ of lacunæ and canaliculi. This part of the tissue consists of a dozen or more rows of lacunæ displayed side by side in due order and regularity. The band of tissue sweeps round the apical space, and diminishes in thickness as this is reached.

The apex of the pulp canal is a little enlarged to give off a devious ill-formed passage into a large, almost globular cavity, which from certain signs of absorption, probably, in the recent state, contained pus.

Round the internal surface of this root the cementum is enormously broad, is exceedingly granular, and possesses in its central

FIG. 93.—Similar to the preceding. Magnified 50 times. D. Dentine; H.C. Hyperplasic cementum, occupying what had previously been the periodontal membrane; O. Osseous tissue.

axis many irregular lacunæ. The neighbouring surface of the posterior root is noted for its thickened cementum, myriads of lacunæ, the canaliculi of which run towards the middle line. The apex and distal portions of the second root present granular cemental matrix, marked granular layer, and abundant lacunæ.

FIG. 94.—Vertical section of radicular portion of a human premolar anky-
losed to the jaw.   Prepared by decalcification.   Stained with hæmatoxylene.
Magnified 10 times.   R. Root of the tooth; A. Bone of jaw.
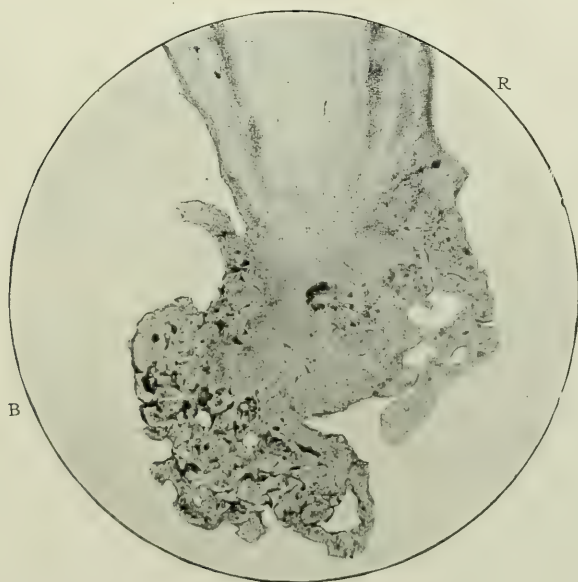
FIG. 95.—Vertical section of a human tooth ankylosed to the jaw.   R. Root;
B. Bone of jaw.   The absolute continuity of the two hard tissues is strikingly
shown.   (*From the collection of the late Storer Bennett.*)

The most external tissue is bone of the alveolus structurally altered. There is little attempt at the regular formation of Haversian systems. The thickest masses contain many elongated spaces filled with soft material, and lacunæ of every conceivable form and size—many of which are abrachiate—abound, some massed together and coalesced, others quite discrete. The matrix is rough and granular. No well-marked osseous lamellæ are visible. The thinner region consists of matrix and lacunæ.

At that portion of the mass furthermost from the roots the tissue begins to assume the characteristic appearances of cancellous bone. The sections are remarkable from the absence of the festooned contours of the foveolæ of Howship. Figs. 91 and 94 are teeth from the mouth of a man aged 41, who was suffering from suppuration of the socket of a tooth which had been recently removed. The second right maxillary premolar and the second and third molars presented complete synostosis of the roots. Fig. 91, from a photograph by the author, shows the general macroscopical appearances of the second molar, which was otherwise normal; and the photomicrograph of low power magnification is that of a decalcified section of the second premolar. Fig. 96 is noteworthy on account of the large amount and size of the bone ankylosed to the root of the premolar.
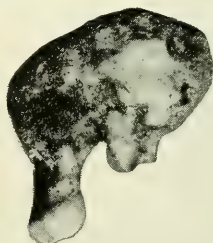
Fig. 96.—Maxillary first premolar ankylosed to alveolar bone.

Fig. 95 is a photomicrograph of one of Storer Bennett's specimens.

(iii) *Hyperplasia*[1]

Definition.—A pathological overgrowth of cementum due to increase in number (not size) of its structural elements. The condition has been called "hypertrophy"[2] of the cementum. This is incorrect, as it is an inflammatory thickening of the tissue. *Synonyms:*—Exostosis, cementosis. *Analogue:*—Osteoplastic periostitis.

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[1] See "A Handbook of Pathological Anatomy and Histology," by Professors Delafield and Prudden, p. 93, 1902.

[2] By a simple "hypertrophy," modern pathologists understand simple increase in size of the elementary structures of a part, leading to structural changes the result of increased nutrition, being dependent on excessive functional activity of the constituents of that part. Thus, the following examples may be cited: hypertrophied bone; enlarged and dilated heart ("compensatory hypertrophy"), congenital macroglossia, enlarged tonsils, etc.

Etiology.—Functional activity of the cells of the periodontal membrane under the influence of the inflammation of chronic periostitis induces hyperplasia. The membrane becomes hyperæmic and swollen, and a small-celled infiltration occurs. On the inflammatory products becoming organised into fibrous tissue, ossification of the inner surface may supervene; but the whole thickness of the membrane does not calcify.

Secondary Changes.—None.

Macroscopical Appearances.—The affection may be so slight as to give rise to no appreciable amount of tumefaction. Ordinarily,

FIG. 97.—Hyperplasia of the cementum. Prepared by grinding. Unstained. Magnified 51 times. (*Photomicrograph by Douglas Gabell.*)

however, smooth rounded nodules may be seen after removal of teeth, or there may be a rough, cancellous, bulbous growth. The whole or part of the root may be diseased; a band or mass of new tissue may not only produce apparent or real elongation of the roots, but actually, in some circumstances, join them or, occasionally, even teeth themselves together. This condition is known as "false gemination" (see previous chapter). In these extreme cases the intervening alveolar septa have been absorbed by the small cells (leucocytes) of the inflamed periodontal membrane.
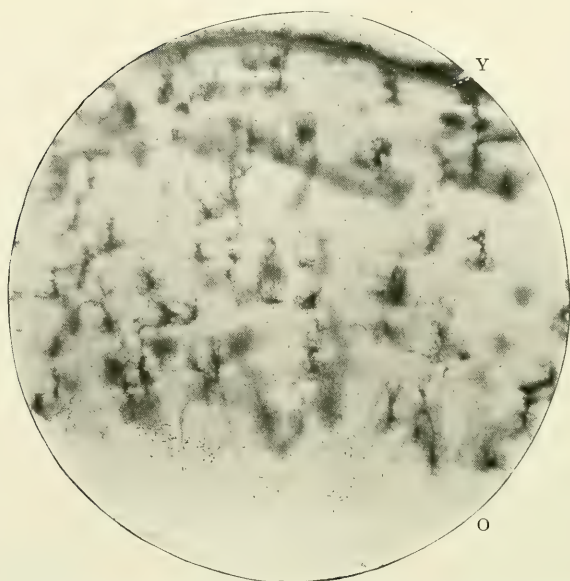
FIG. 98.—Hyperplasia of the cementum. Magnified 150 times. o. Oldest, or the earliest deposited layer; y. Youngest layer.
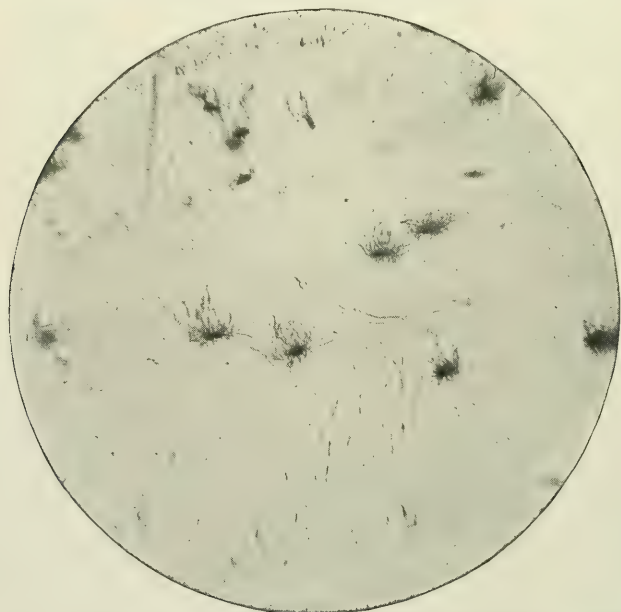
FIG. 99.—Lacunæ and canaliculi in hyperplasic cementum. Prepared by grinding. Unstained. Magnified 170 times. (*Photomicrograph by Douglas Gabell.*)

To the naked eye the root membrane appears considerably thickened and reddened.

Secondary Changes.—None.

The microscopical structures vary in different degrees. Hyperplasia may be limited to the apex of a root, a minute portion near the enamel, or midway down the side of the root.

The chief features of the new growth, in addition to the amorphous or granular matrix, lamellæ, and perforating and penetrating fibres, are lacunæ and canaliculi. It has generally been understood these latter bodies exist in normal cementum; but, as has already been pointed out in Chapter V., Vol. I., this is most probably an erroneous idea. It is appropriate and opportune, therefore, to describe cemental lacunæ in this place.

The lacunæ somewhat resemble those of bone (*q.v.*): but in their form, number, size, arrangement and extraordinary length and number of their processes, they present several points of difference.

A cemental lacuna is an irregular space. In dried sections it is filled with *débris* or air, in the fresh state, with some amount of living material. It may be rounded, ovate, plumiliform, arborescent or rimous in shape. The first two conditions most generally obtain. The lacunæ of bone are much more regular.

In number, these bodies vary in different parts. Thus at the cervical margin they are generally absent. Few in number, and isolated in position, they are found midway between the crown and the root; while in the apical region they are most abundant. In man, the thinnest cementum is almost devoid of them; the thickest is always accompanied by them.

Lacunæ differ in size. According to Kölliker, they may range in diameter from 10μ to 40μ or 60μ. These are average measurements. They are larger than those of bone. Those having the greatest size are to be found in the inter-radicular regions of molar teeth, and also wherever the cementum is thickest (see Figs. 102 and 109). There is no definite arrangement of their position as in compact bone; they are placed in a haphazard fashion throughout the matrix, though sometimes they lie in series, apparently being governed in their distribution by the lamellæ. Their processes are numerous ramified channels, commonly known as canaliculi, which stretch out for some considerable distance (sometimes as far as 6μ or 8μ) from

the lacuna, to which they very often give a jagged plumiliform appearance (see Fig. 101). Branched and irregular in outline, as a rule, these offshoots taper towards their free ends where they commonly measure 1μ. Some end in a *cul-de-sac;* others anastomose with neighbours; others again form the minute extremities of the

FIG. 100.—Hyperplasia of the cementum. Prepared by grinding. Stained by impregnation with coloured collodion. Magnified 200 times. Shows the diversified character of the lacunæ.

dentinal tubes or their branches, while another set may meet with certain processes from the spaces of the granular layer of Tomes. In slight cases of hyperplasia it seems to be a normal condition for the canaliculi to be given off from all sides of the lacuna; still there is often a general disposition on their part to most chiefly extend
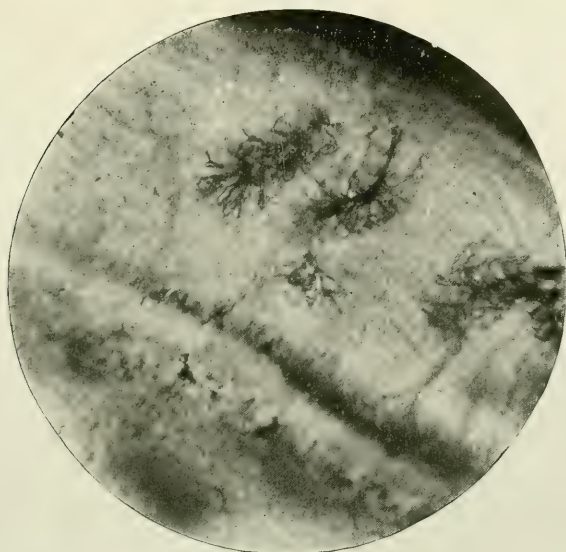
FIG. 101.—Plumiliform lacunæ in hyperplasic cementum. Prepared as in the preceding figure. Magnified 250 times.
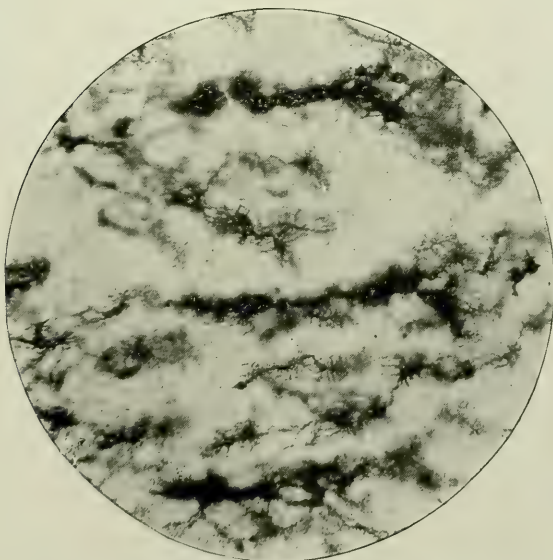
FIG. 102.—Rimous and arborescent lacunæ in hyperplasic cementum. Prepared as in the preceding figure. Magnified 250 times.

outwards towards the periodontal membrane. In rimous lacunæ they issue at right angles to the sides as well as the ends; and, in addition, in the innermost zone—viz., that first laid down by osteoblasts, the outlines of the lacunæ when present are very irregular, their canaliculi are short and may blend intimately with many elongated canals. In multi-rooted teeth, where cementum is
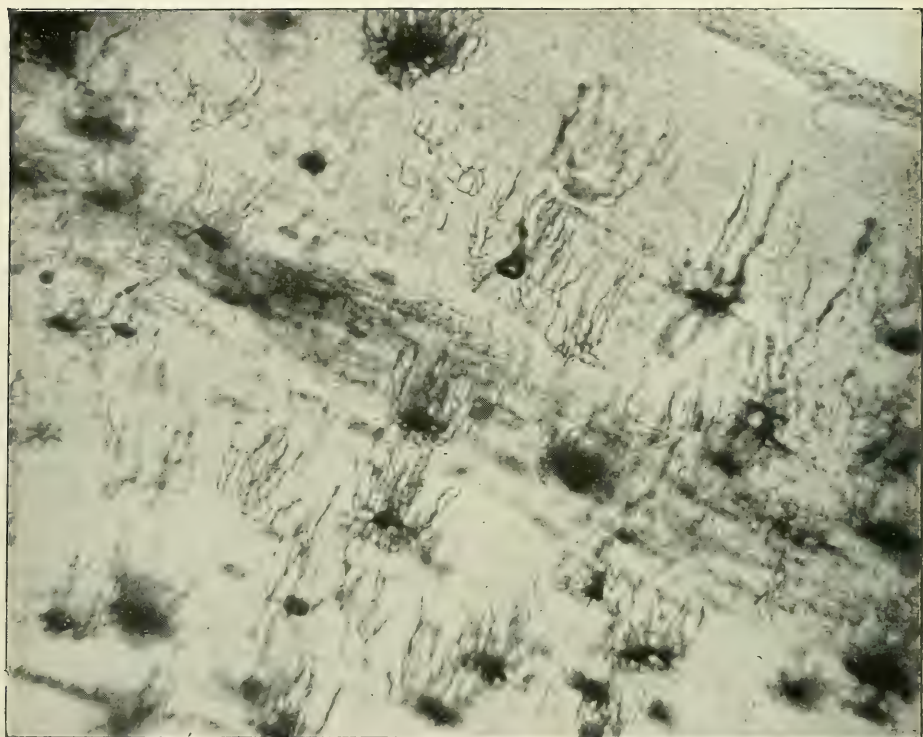
FIG. 103.—Cemental lacunæ and canaliculi. Magnified 1,000 times.
(*Photomicrograph by Norman Broomell.*)

slightly thicker than elsewhere, the ground substance is particularly granular; if lacunæ are present, they are fairly evenly distributed throughout, and many rimous or aborescent spaces are found (see Fig. 109).

According to Tomes (*op. cit.* p. 117), some lacunæ have short canaliculi, and are encapsuled. "Sometimes a line is seen to be surrounding a single lacuna, sometimes several are enclosed within it." Capsules are not very common; but, if present, they appear clearly

defined, are yellowish in colour, have slightly curved borders, and may surround an individual or group of lacunæ either partially or entirely.

What constitutes the actual contents of these spaces in the fresh state has been up to the present time only imperfectly known.   Most probably, arguing from the sensitiveness of this tissue when exposed, each is filled with protoplasm.   Heitzmann and Bödecker both claim to have proved this as a physiological and histological fact.

And it is a common experience to find, in sections of hyperplasic cementum, when properly prepared, lacunæ containing a nucleated
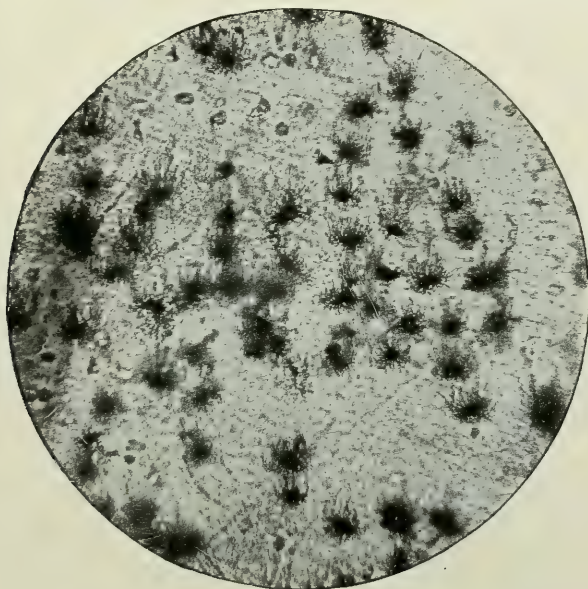
FIG. 104.—Cemental lacunæ in the first-deposited layers of cementum.   Magnified 80 times.   (*Photomicrograph by Norman Broomell.*)

cell, analogous most probably to the bone corpuscles of ordinary osseous tissue.

In addition, the incremental lines of Salter, generally speaking, are strikingly exhibited in ground as well as in decalcified sections. They are not, however, always constant, as in Fig. 105.

If the deposition of the tissue has been conducted on uniform iines, the laminæ are very regular, the lacunæ, though numerous, bear also some definite relationship to each other, and are systematically arranged with regard to the laminæ (see Fig. 107).   But when the
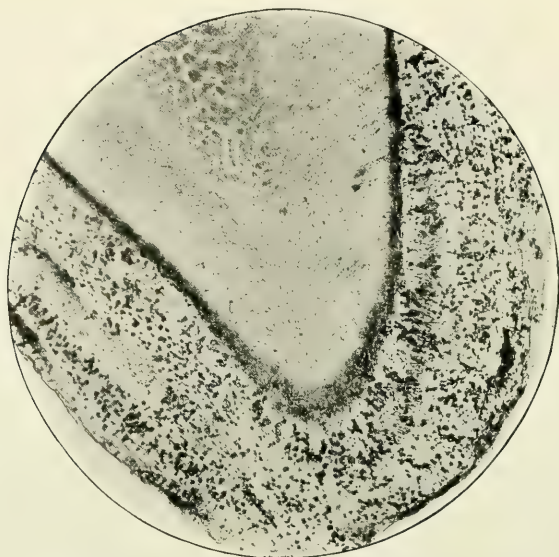
FIG. 105.—Hyperplasia of the cementum. Prepared by grinding. Unstained
There are no incremental lines. Magnified 40 times.

FIG. 106.—Hyperplasia of the cementum. Transverse section through apical
region of tooth. Magnified 40 times. D. Dentine; R.C. Root canal somewhat
enlarged and containing a deposit of ossific or calcific material; H.C. Hyperplasic
cementum. (*From a specimen in the collection of Douglas Caush.*)

FIG. 107.—Hyperplasia of the cementum. Magnified 40 times. D. Dentine;
H.C. Cementum in which the laminæ are regularly arranged; V.C. Vascular canal.
(*From the collection of Douglas Caush.*)

FIG. 108.—External absorption of dentine and deposition of hyperplasic
cementum. Magnified 45 times. D. Dentine; A. Area of absorption; H.C.
Cementum. (*From the collection of Douglas Caush.*)
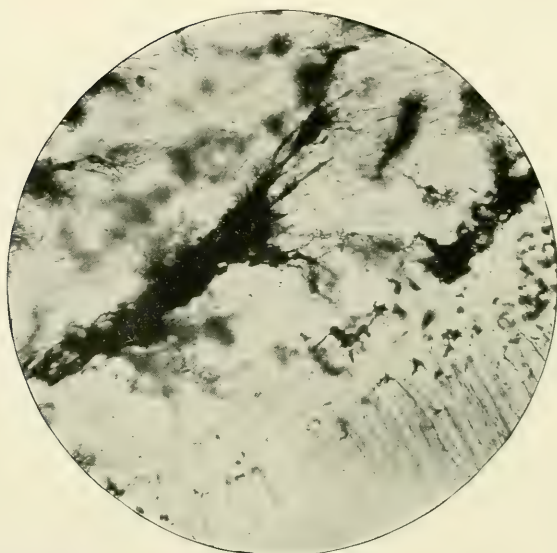
FIG. 109.—Rimous and very large lacuna in hyperplasic cementum. Magnified 45 times. (*From the collection of Douglas Caush.*)

FIG. 110.—Internal absorption of dentine, enlargement of root canal, and deposition of osseous or calcific material. Magnified 40 times. D. Dentine; R.C. Root canal; O. New tissue deposited in the root canal; H.C. Hyperplasic cementum. (*From the collection of Douglas Caush.*)
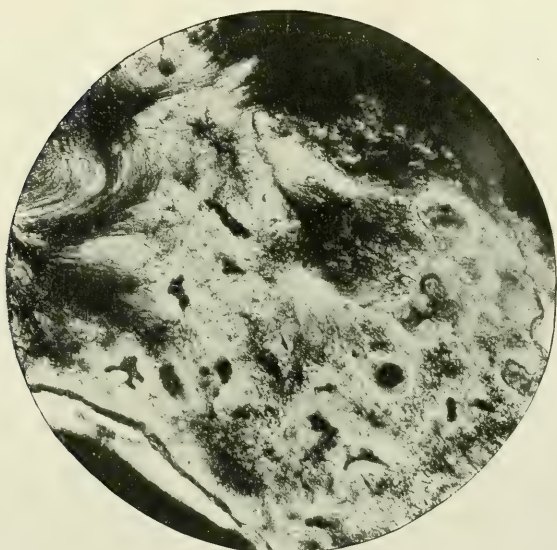
FIG. III.—To show the nature of the new tissue deposited in a root canal Magnified 250 times. (*From the collection of Douglas Caush.*)
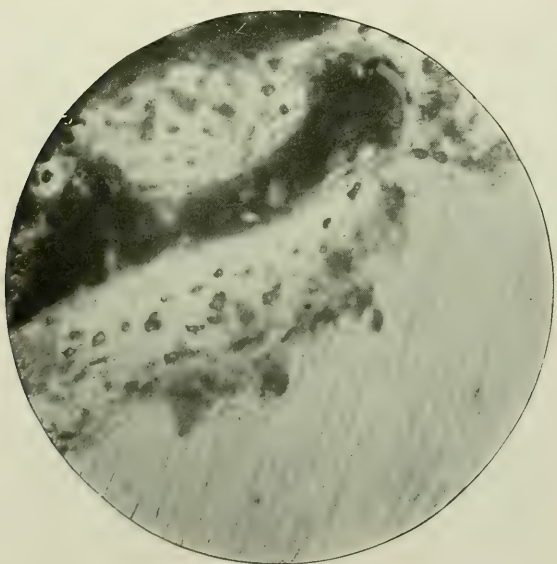
FIG. 112.—External absorption of dentine, and deposition of hyperplasic cementum in the absorption areas. Magnified 200 times. (*From the collection of Douglas Caush.*)

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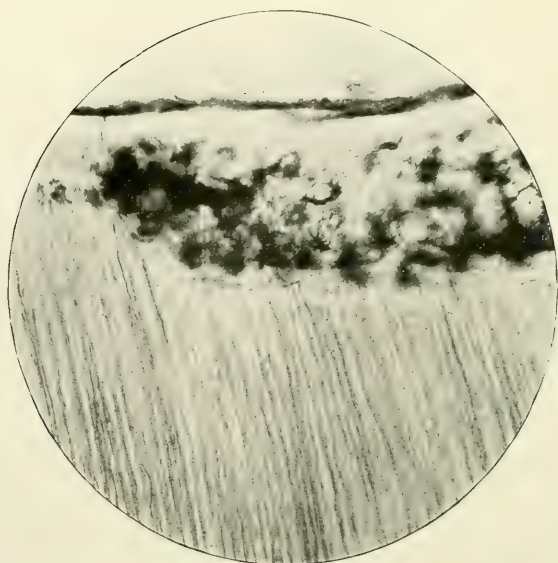
FIG. 113.—Similar to the preceding. From the same source. Magnified 200 times.
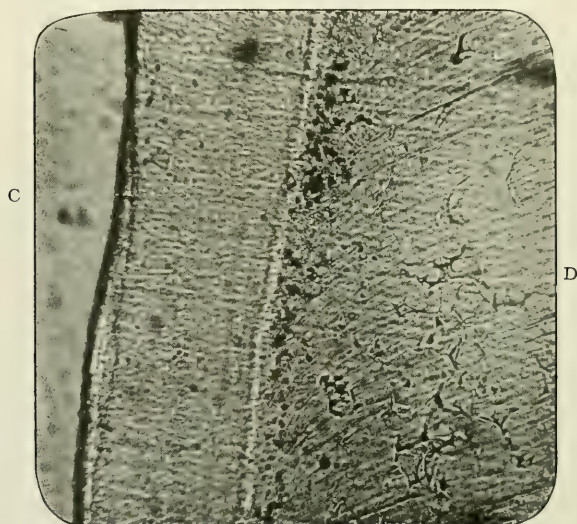
FIG. 114.—Cementum and dentine of a senile tooth. Prepared by grinding. Unstained. Magnified 230 times. C. Granular, but not lacunated cementum; D. Dentine.

mass is lobulated and has a rugged surface, then the lacunæ are coarse, large, and provided with long branched deviating processes. In the former degree the line of junction with the dentine is sharp and clear; in the latter it is often impossible to say exactly where one tissue ends and the other begins.

FIG. 115.—Senile cementum and alveolar bone. Magnified 45 times. D. Dentine; C. Hyperplasic cementum; P.M. Periodontal membrane; B. Osteoporotic alveolar bone.

Newly formed vascular channels often make their appearance in the cementum (Fig. 107) and the phenomena of absorption and re-deposition (Figs. 112 and 113) take place side by side in the same specimen. The condition is commonly associated with enlargement of the pulp canal, and sometimes depositions of new osseous material (see Figs. 106 and 110).

(iv) *Senile Cementum*

Of cementum found in aged teeth little need be said. As a rule, it is hyperplasic, because few teeth which have been removed from the mouth, either from mobility or to relieve pain, have escaped the ravages of disease, and their cementum is therefore thickened and
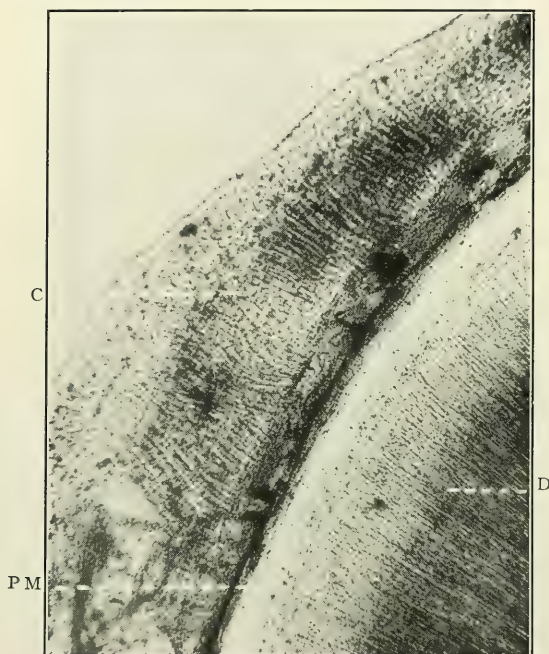
FIG. 116.—Senile cementum. Magnified 200 times. D. Dentine; P.M. Homogeneous layer; C. Cementum.

lacunated. But healthy teeth, which have never been the subject of an attack, show that this tissue is somewhat thicker than in young adults, but is structureless and similar in every way to normal cementum (see Fig. 114. *Cf.* Fig. 97).

CHAPTER IV

DENTAL CARIES

Microscopical Elements in:—(i) Nasmyth's membrane; (ii) "White spots;" (iii) Zones of partial and complete decalcification; (iv) "Secondary enamel decay;" (v) Zone of translucency; (vi) "Liquefaction foci;" (vii) "Pipe-stem" appearance; (viii) Opaque zones of Miller; (ix) Cementum; (x) "Arrested" caries.

Definition.—"An acid fermentation during which the inorganic portions of teeth are first dissolved, and then the organic portions disintegrated by the action of micro-organisms." More shortly:—Decalcification of the teeth followed by their dissolution.

Etiology.[1]—The environment of the teeth. The peptonising influences of micro-organisms on the surface, proceeding inwards. Strictly it is not a disease, as it cannot be induced at will, in man or the lower animals.

Dental caries has never been noted in the teeth found in ovarian teratomatous cysts.

Macroscopical Appearances.—These are too well known to require any special description; suffice to say that enamel loses its polish; white, opaque "spots" appear; pigmentation ensues; and in the hard parts, cavities are produced. According to certain accompanying phenomena it has been termed *caries acuta*, when rapid in progress; *caries chronica*, when slow; *caries sicca*, when dry; and *caries humida*, when saturated with fluids. French writers speak of caries of the "1st degree," "2nd degree," and so on, thus hinting at the extent which has taken place.

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[1] The causes of Dental caries, it may be stated in general terms, are not altogether known. Some authors have divided the causes into (*A*) Predisposing, (*B*) Exciting. Of these, grouped under the heading of General predisposing causes, one finds, (*a*) Heredity, (*b*) Improper dietary, (*c*) Civilization; and under the heading of Local predisposing causes, (*a*) Malposition in the dental arches, (*b*) Structural defects of the surface, and (*c*) Vitiated oral secretions, produced by (i) systemic diseases, *e.g.*, gout, typhoid fever, etc.; (ii) occupation, as in millers, or finally, (iii) pregnancy. The Exciting cause is: Bacterial fermentation of carbo-hydrates. It is difficult to name the true cause or causes. Probably a hitherto undiscovered special caries-producing micro-organism is the chief agent.

Secondary Changes.—The condition may become arrested; but usually it extends, unless interrupted, until the crowns of teeth are destroyed more or less in their entirety.

Phenomena.—The probable sequence of events in progressive caries of the teeth is as follows:—

1.—Attack on Nasmyth's membrane,
2.—Formation of "white spots" in enamel,
3.—Channeling and penetration of enamel by micro-organisms,
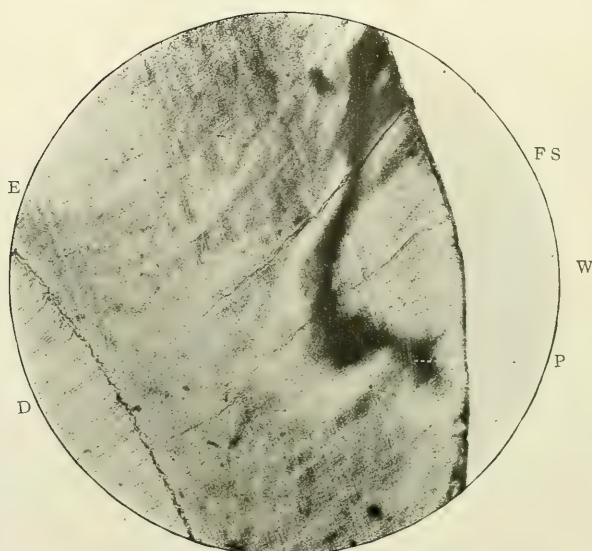
FIG. 117.—An early stage of caries of the enamel. Prepared by grinding. Unstained. Magnified 45 times. E. Enamel; D. Dentine; W. "White spot;" P. Area of pigmentation; F.S. Free approximal surface of the tooth.

4.—Decalcification of dentine,
5.—Tubular infection by micro-organisms, with formation of "liquefaction foci,"
6.—Fusion of the latter and production of cavities, and
7.—Dissolution of the hard parts.

These must now be severally described.

HISTOLOGY

(i) *Nasmyth's membrane*

Of the histo-pathology of this tissue little is known. Its extreme tenuity and its anatomical characteristics, and the difficulty of

making sufficiently satisfactory microscopical preparations have given but few opportunities for pathologists to work out its morbid anatomy.

. It would appear that the tissue is often more or less deeply pigmented, probably partly through the agency of certain chromogenic bacteria, partly through the use of tobacco, or the salts of iron, copper and mercury, and partly through *post mortem* changes common to all organic matter; and finally wholly or partially removed by the action of micro-organisms and mechanical agencies from the surface
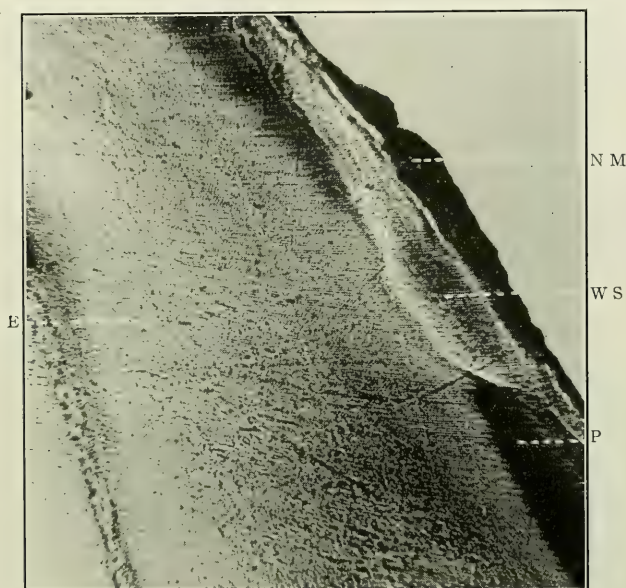
FIG. 118.—Early stage of caries of enamel. Magnified 90 times. N.M. Nasmyth's membrane increased in thickness (? carious); w.s. "White spot;" P. Pigmentation of enamel; E. Non-carious enamel.

of the enamel. It is believed that the natural acids of fermentation have little or no effect upon it, since the translucent pellicle is peculiarly resistant to strong acid reagents like hydrochloric, acetic, etc. It is, however, evident that the enzymes of bacteria are able to disintegrate this film with, probably, the production of a small amount of amino-acetic acid. In preparations stained by Gram's method, it is possible to see masses of micrococci congregated on its surface.
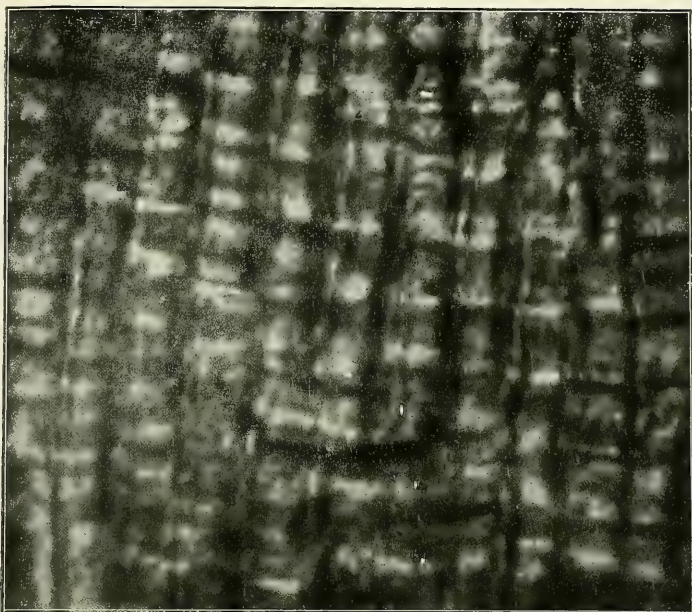
FIG. 119.—A portion of a "White spot."   Magnified 2,000 times.   (*Photomicrograph by Leon Williams.*)
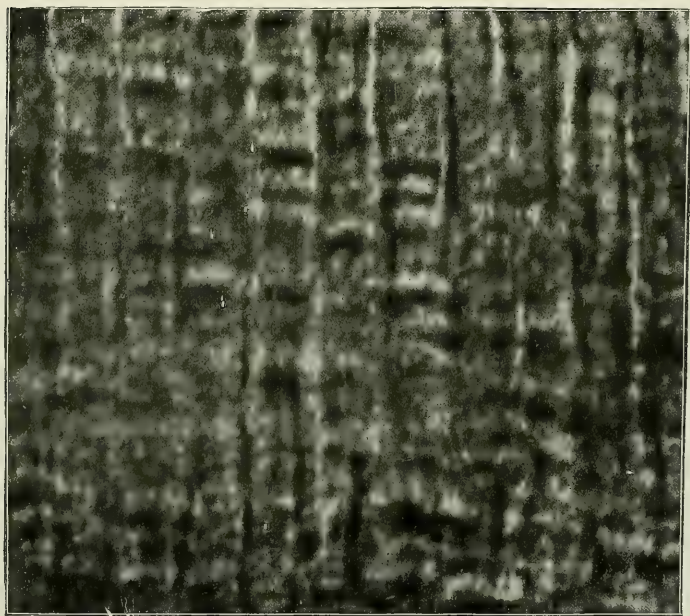
FIG. 120.—Section through human enamel, showing the first stages of caries. Magnified 2,000 times.   (*Photomicrograph by Leon Williams.*)

Otto Walkhoff in a recent work[1] describes and figures, in Plate XI., a deposit of green pigment which has taken place, in parallel rows, between the "convexities of the enamel." This, no doubt, refers
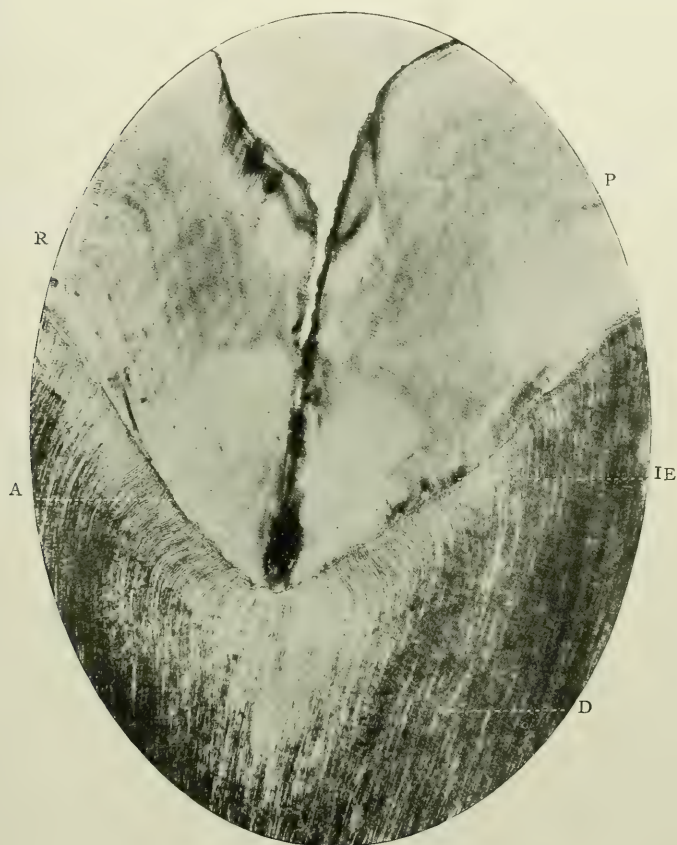
FIG. 121.—An early stage of caries of the enamel round the fissure on the occlusal surface of a human premolar. Prepared by grinding. Unstained. Magnified 45 times. P. Carious patch at free end of a deep sulcus; I.E. Developmentally defective enamel rods; R. Brown striæ of Retzius; A. Amelo-dentinal junction; D. Dentine.

to the thickest portions of the membrane in the pits and crevices of the enamel.

It may, however, be stated that the precise rôle played by Nasmyth's membrane with regard to dental caries is not at present

[1] "Mikrophotographischer Atlas der Pathologischer Histologie Menschlicher Zähne."

understood.   It would seem, however, in the opinion of the author
to constitute, under favourable conditions, the first line of defence
against dental caries.   Pickerill, and Tomes and Nowell ("A System
of Dental Surgery," 1906) present the hypothesis that it may act as
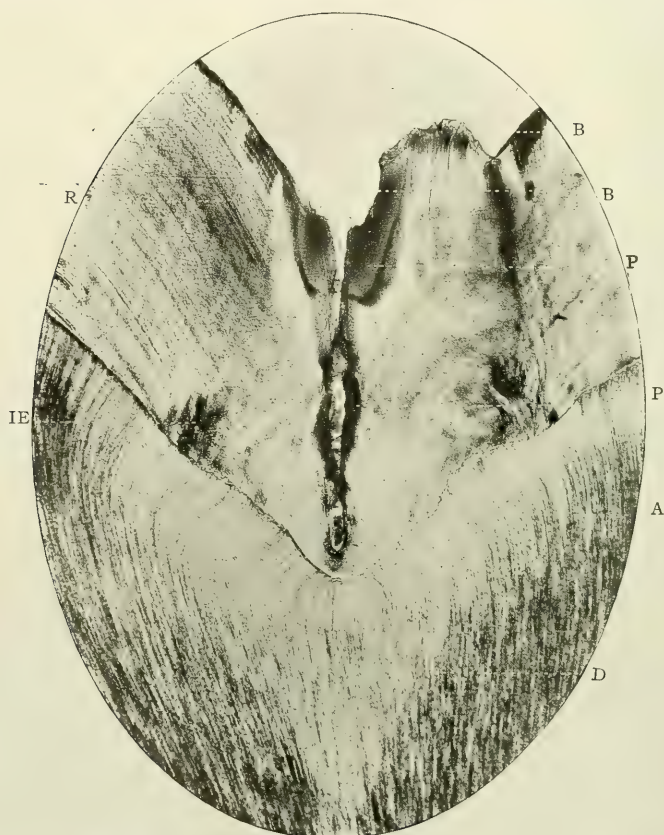
FIG. 122.—Similar to the preceding, but caries more advanced. Prepared
similarly, with same magnification. Lettering as in preceding figure. **B.B.**
Breach of surface.

a dialysing membrane to certain substances, which may disintegrate
the surface of the enamel and lead to carious changes in that tissue.

(ii). "*White Spots*"

Quite dark by transmitted light, and often surrounded by a
pale zone, Fig. 117, these have probably the same histological

features as that surrounding the pigmentation areas in caries. The enamel rods in the "white spots" are exceedingly granular, and deeply pigmented, their enamel globules, according to Leon Williams, having become but imperfectly fused owing to insufficiency of cement-substance (Figs. 119 and 120). Very light and deeply pigmented patches of enamel alternate irregularly in the immediate vicinity of the "white spots."
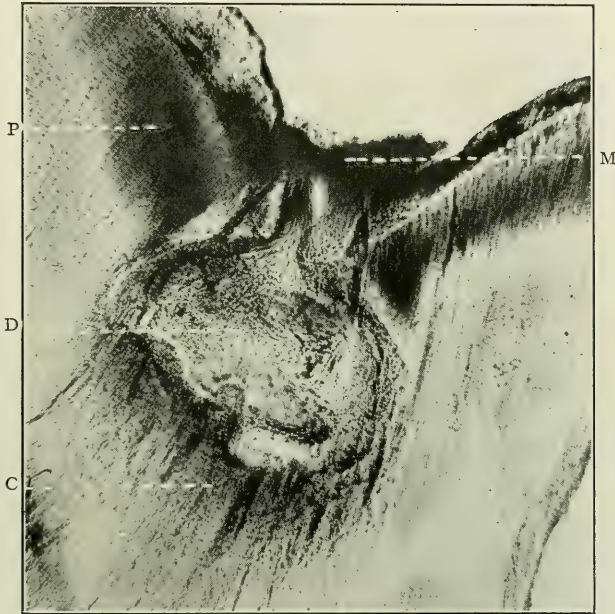
FIG. 123.—Caries of enamel. Magnified 90 times. M. Micro-organisms; P. Pigmentation of enamel; D. Decalcified enamel; C. Decalcifying enamel.

(iii) *Channeling by Micro-organisms*

The appearances produced by the earliest action of the acids generated by the bacteria upon enamel vary. This is due to differences in the structure of the tissue as well as to the variations in the products or peptonising effects of the micro-organisms themselves. (Leon Williams.) (Figs. 127 and 128.)

It is still an undecided question whether the "gelatinous plaques" of Black[1] and Leon Williams do really exist. They are believed to be formed by masses of bacteria which, collecting in suitable

[1] Black, G. V.: "Operative Dentistry," Vol. I, 1908.

situations, are able by a precipitation of mucilaginous compounds to focus in a more or less widespread but yet well-defined fashion the operations of the destructive agencies. Kirk[1] has recently apparently shown that it is possible to manufacture "bacterial plaques" to any size or extent (Figs. 126 and 127). The author, however, believes that these "gelatinous plaques" are in reality nothing more nor less than portions of Nasmyth's membrane which have been retained *in situ*. For the free surface of such a membrane
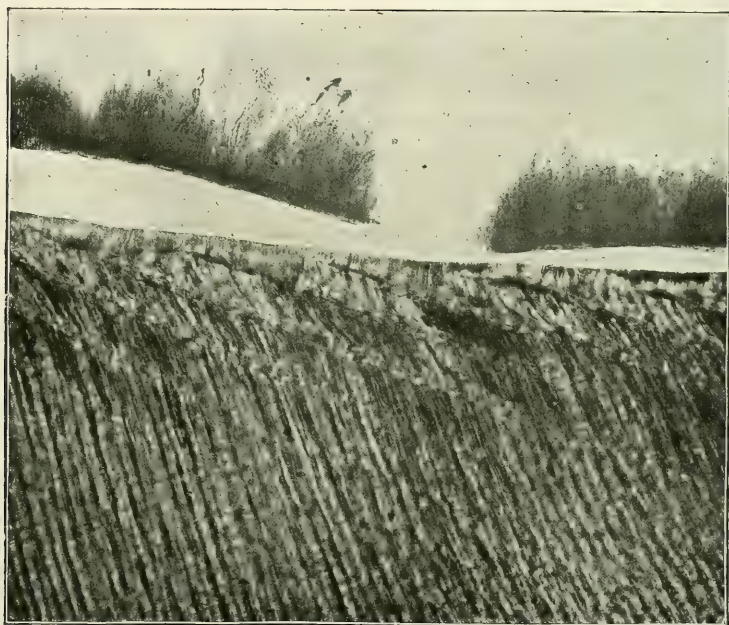
FIG. 124.—Section of normal human enamel. Magnified 350 times. It shows a thick, felt-like mass of micro-organisms slightly raised from the surface of the tissue (Nasmyth's membrane) produced by pressure of the cover-glass in mounting. (*Photomicrograph by Leon Williams.*)

could afford attachment for material which might become a suitable nidus for the development of oral bacteria.

The acid dissolves out channels, or "wells," whose walls may be parallel or V-shaped (Fig. 126). These are most commonly seen on the approximal surface of teeth of man; their occurrence in the buccal or lingual aspects being less frequent. They penetrate

[1] Kirk: "A Consideration of the Question of Susceptibility and Immunity to Dental Caries," *Dental Cosmos*, 1910.

the intercolumnar substance between the rods, not the rods them-
selves (Figs. 125 and 126). They are often larger than the di-
ameter of a rod, and are in no sense of the word developmental
defects, but merely produced by the acids formed by the enzymes of
the micro-organisms of caries.
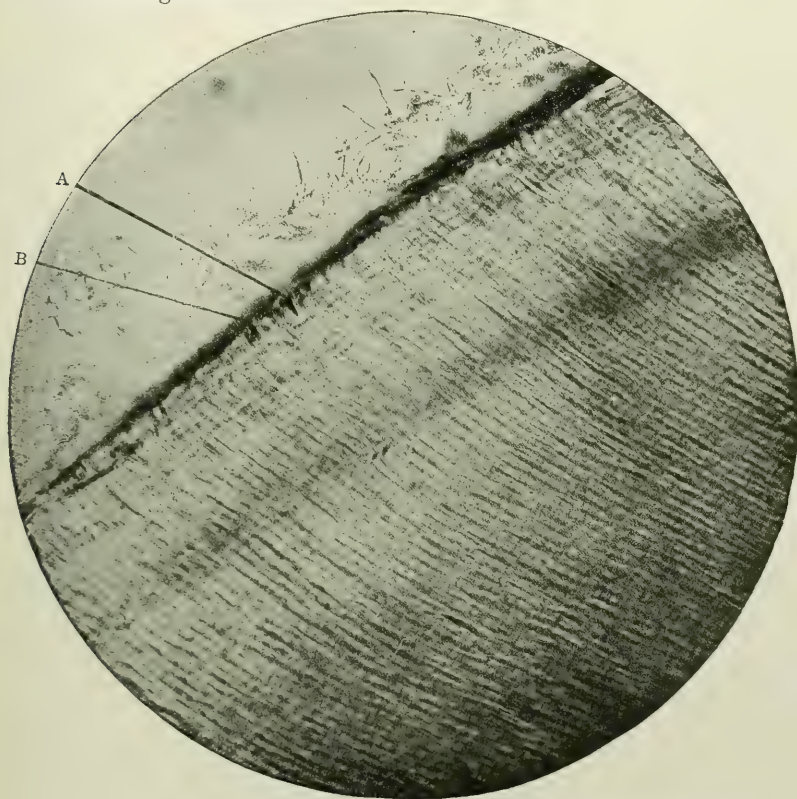
FIG. 125.—A section of human enamel showing micro-organisms attached to
the approximal surface of a molar tooth. Magnified 200 times. A.B. The
commencement of the process of caries by solution of the cement-substance.
(*Photomicrograph by Leon Williams.*)

The acid passes inwards for some distance by means of these
channels without the production of discolouration.

Dark bands in enamel, internal to the line of acid penetration,
probably represent a partial arrest in the action of the micro-
organisms, which has been presumably brought about by some
altered conditions of the environment of the tooth (Fig. 132).

In acute caries the acid penetrates quickly and deeply along

the lines of least resistance, *i.e.*, the cement substance, the rods themselves undergoing isolation, and becoming disintegrated before their morphological characters are lost.   In chronic cases, however, the whole substance of the rods is permeated by acid, the result being the appearance of a "sponge-like structure," giving the effect of
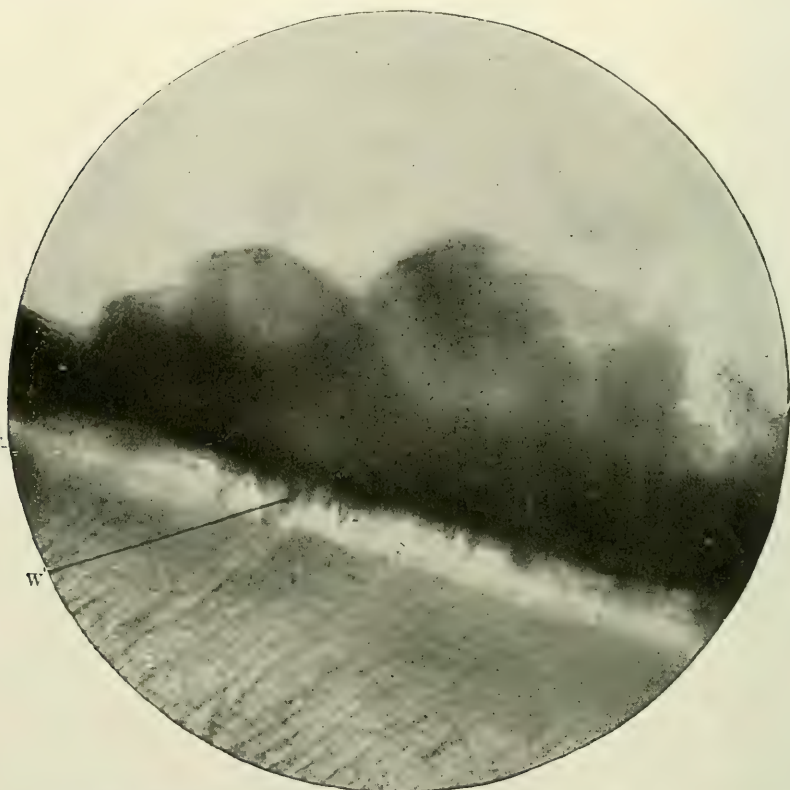
FIG. 126.—Section of human enamel.   Magnified 350 times.   It shows a deeply stained mass of micro-organisms attached to the surface, and, at w, further action of the acid in dissolving the cement-substance and forming V-shaped spaces between the enamel rods.   (*Photomicrograph by Leon Williams*.)

discolouration in sections when viewed by transmitted light, and chalky white by reflected light (Leon Williams).

Thus, acute white caries is unaccompanied by discolouration, and is due to solution of the intercolumnar substance; chronic caries to impregnation with the products of bacteria and associated with marked discoloration.   These changes are macroscopically invisible, and occur before a breach of surface results.

With regard to congenital defects in enamel, it is almost con-
clusively proved that they are not favourite sites for the development
of caries; but that the micro-organisms most frequently act on enamel
which is *not* over the cusps of teeth, where it is usually most defec-
tive.   Caries often begins, as is well known, from a clinical stand-
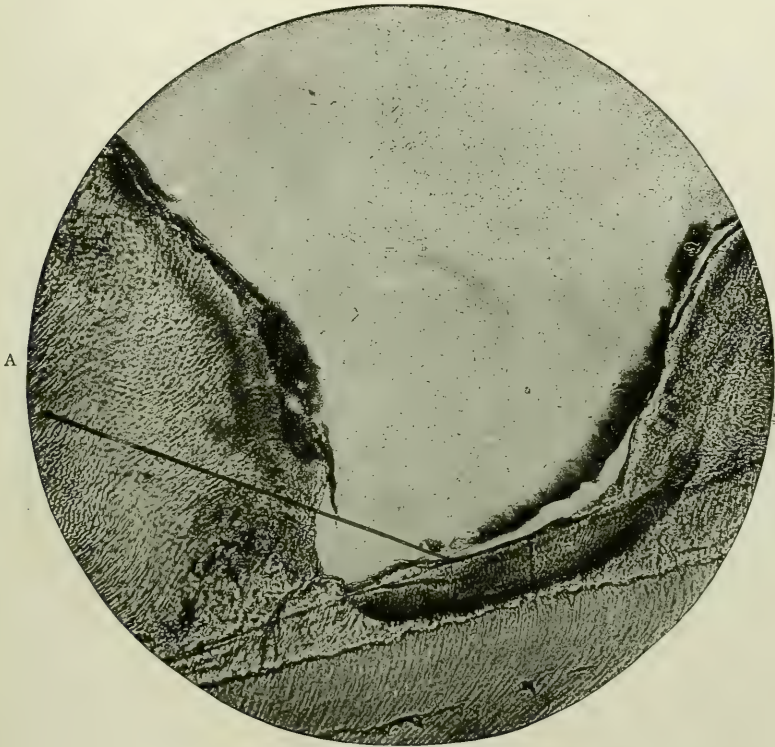
FIG. 127.—A section of human enamel from the approximal surface of an
incisor.   Magnified 175 times.   Showing enamel nearly penetrated by dental
caries.   A. The carious process apparently arrested by a line of stratification.
Micro-organisms (attached to Nasmyth's membrane) slightly raised from floor
of cavity by shrinkage caused by mounting in balsam.   (*Photomicrograph by
Leon Williams.*)

point, on approximal surfaces of the teeth, and here the enamel is,
probably, more perfectly formed than elsewhere.   But, while bac-
teria act independently, there is no doubt that they avail themselves
of the presence of pits and fissures, being governed in their disposi-
tion on the surface of enamel by the completeness or incompleteness
of the translucent pellicle of Nasmyth's membrane.   The explana-

tion would appear to be, that having once penetrated through an abraded edge of the membrane they can pass beneath it to some considerable distance. If this is true, the phenomena of caries beginning in pits and fissures would be accounted for.

At a somewhat later stage, carious enamel, under the microscope, shows that the individual rods are pronounced, their intercolumnar
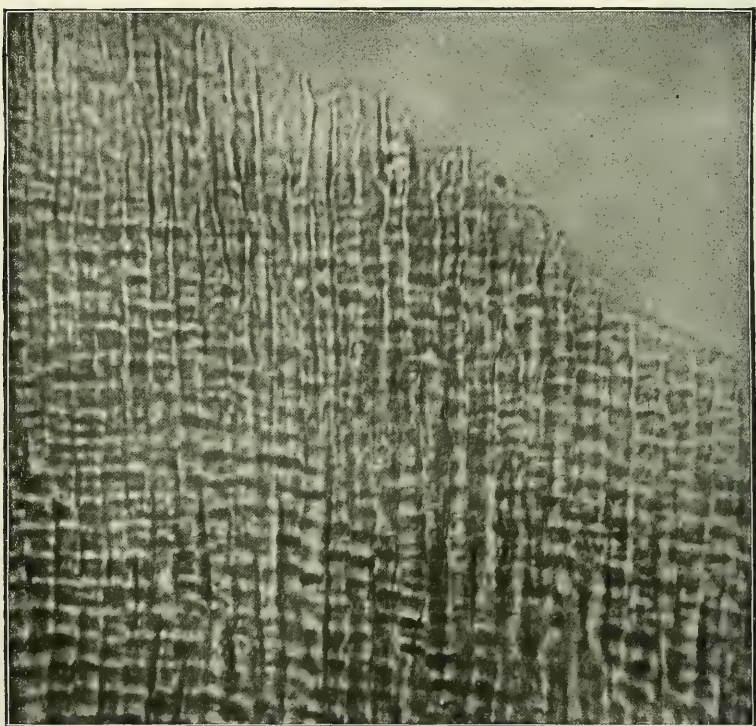
FIG. 128.—A section of carious enamel. Magnified 550 times. The bacteria have been removed to shew the action of the acid on the enamel rods. (*Photomicrograph by Leon Williams.*)

substance partially loosened (Figs. 128 and 130), and clearly exhibiting spaces between the enamel columns. The striæ are very marked. This is on the surface. Lower down, the structure is indistinguishable, the rods are confluent, and have lost their outline; they appear exceedingly granular. Still deeper, they have disappeared, their place being occupied by masses of micro-organisms, which are clearly revealed when the section has been stained with
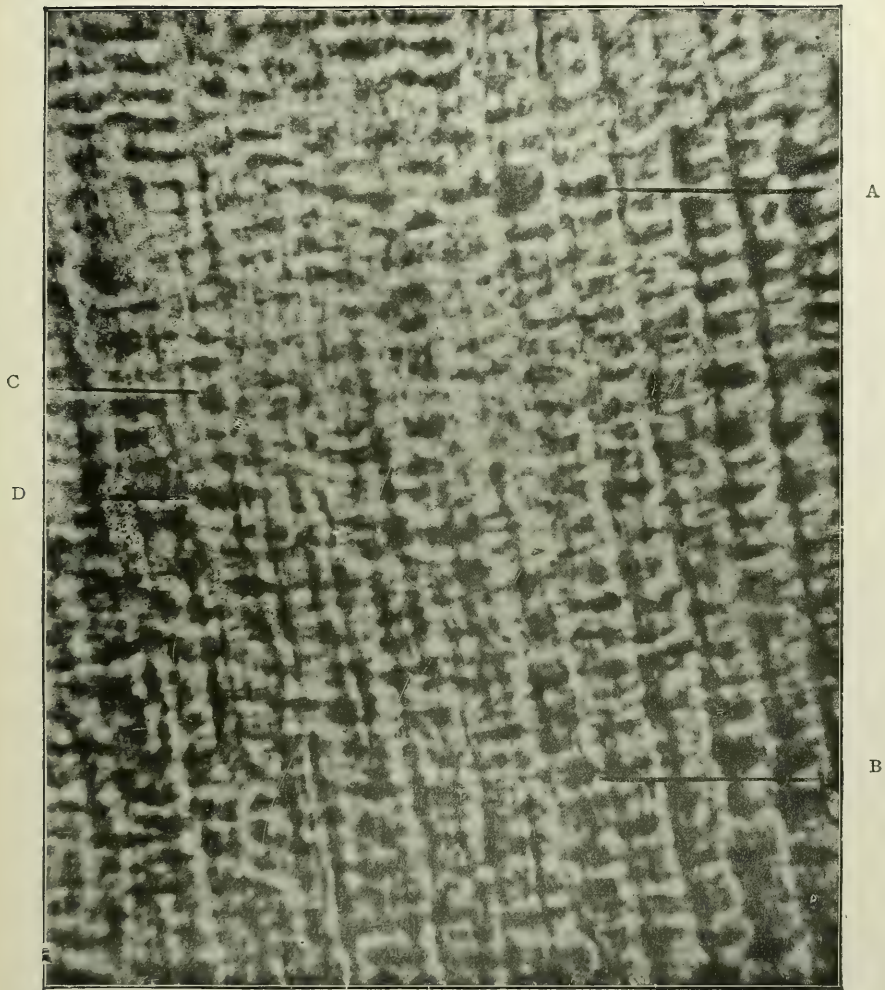
FIG. 129.—A section of carious enamel, showing appearances of the tissue in a case of chronic caries. Magnified 2,000 times. The cement-substance has been dissolved away, thus exposing to view the original organic matrix. The globular bodies or sections of the rods are shown at A, B, C, D, and are seen to be identical in general shape and character with those originally formed in the ameloblasts and on the surface of forming enamel. (*Ph. tomicrograph by Leon Williams.*)

8

gentian-aniline-violet, fuchsine, or other aniline dyes. Bacteria may be seen occupying the spaces between the rods if a minute fragment of "secondary enamel decay" is examined.

The loosening of the rods is said to be dependent on the action of lactic acid. Tomes believes that the axes of the enamel columns

FIG. 130.—A cover-glass preparation from scrapings of white, opaque carious enamel. Magnified 450 times. The cement substance between the rods is seen to be dissolved away, and the crevices thus formed are filled with round and rod forms of micrococci and bacteria. Stained by Gram's method. (*Photomicrograph by Leon Williams.*)

are attacked before their peripheries. This, no doubt, is the case where the tissue is at all ill-formed.

Hence there are three zones or areas in enamel which show different appearances:—

(i) The zone of partial decalcification, where the rods are individually distinct, the striæ marked, and the cement substance partially disintegrated,

(ii) The zone of complete decalcification, in which the rods are
   structureless, but possess a marked degree of granularity;
   And

(iii) The zone of "secondary enamel decay," in which masses of
   micro-organisms only can be demonstrated (see Fig. 139).
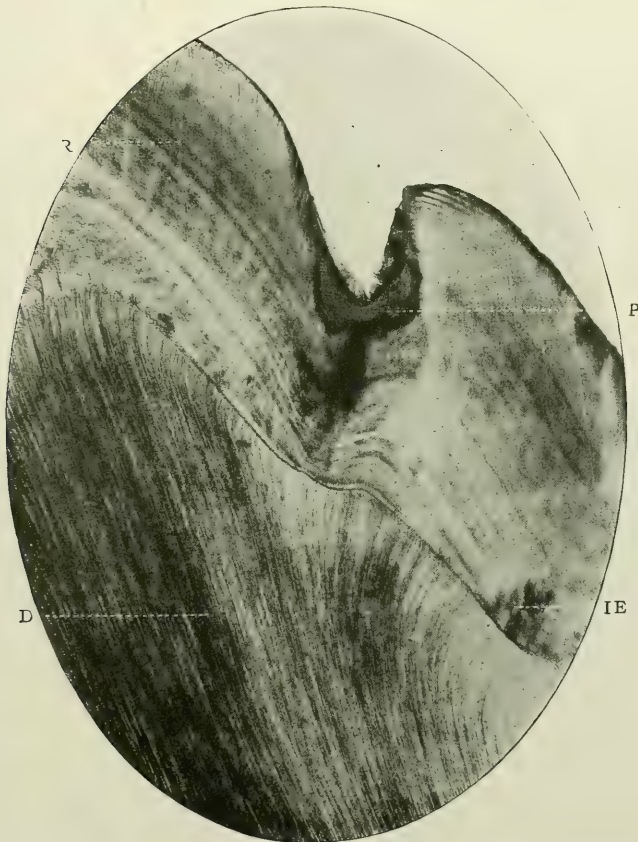
FIG. 131.—Caries of the enamel, having produced a breath of surface. Pre-
pared by grinding. Unstained. Magnified 45 times. P. Carious patch in
enamel; I.E. Imperfect enamel rods; R. Striæ of Retzius; D. Dentine.

(iv) *Decalcification of Dentine*

This invariably, in a fairly regular line, precedes the bacterial
infection of the tubes. The dentine stains easily with histological
reagents. Unstained it is pigmented, being pale yellow or brown in
colour.

A microscopical appearance of great importance in the region of
advancing decalcification is the presence of the "*zone of translucency*"
(Fig. 137).

The term is applied to an area of increased transparency in that
part of the dentine which is situated between the advancing caries

FIG. 132.—A further stage of the preceding figure.   Prepared similarly, and the
same lettering.   Magnified 25 times.

and the healthy tissue.   In longitudinal sections it often appears
conical in shape, the apex being towards the pulp, the circular base
towards the periphery of the dentine.   The shape of the figure is,
no doubt, governed by the convergence of the dentinal tubules,
which radiate from the pulp outwards in a centrifugal direction.   It

may present the character of hyaline stripes or spots; and apparently
owes its existence to the close approximation of the index of refrac-
tion of the dentinal tubules and their contents in this situation,
to that of the matrix of the surrounding dentine.

Amongst the various phenomena of dental caries it would be
difficult to find any of greater interest or upon which opinions still
differ more than the nature of this "translucent zone." It is not
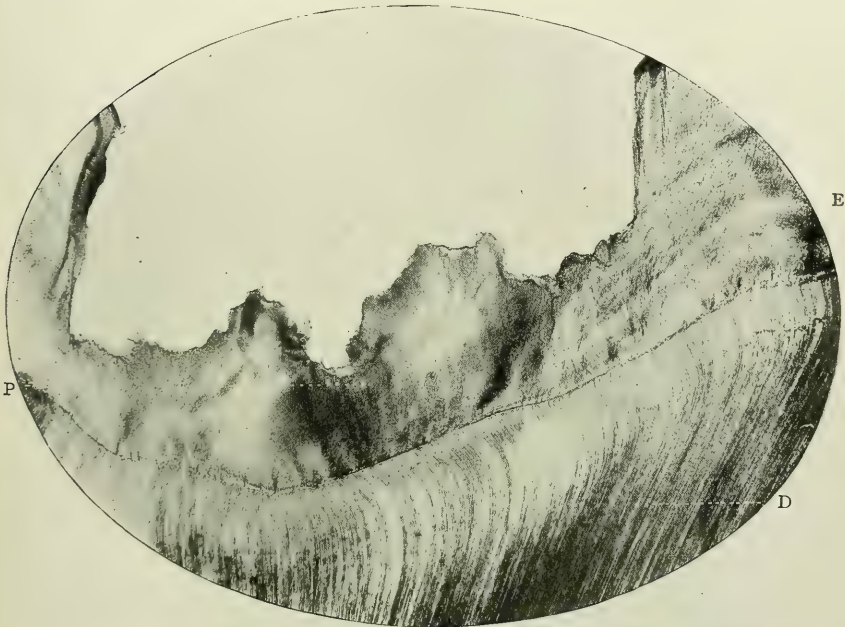
FIG. 133.—A further stage of caries of the enamel. Similarly prepared and
lettered to the preceding. Magnified 45 times. The large cavity is easily
recognised.

always found in carious dentine, and sometimes it exists in places
where there is not a trace of the condition.

Two main opinions are still held regarding its production. Thus,
Sir John Tomes and Miller,[1] with Magitot,[2] and others, considered
it due to vital action on the part of the dentine, which leads to a
calcification of the dentinal fibril.

[1] "Micro-organisms of the Human Mouth," 1889.
[2] "Traitè de la Carie des Dents," 1867.

And secondly Wedl,[1] Black,[2] Leber and Rottenstein,[3] C. S. Tomes, F. J. Bennett,[4] and Abbott, regard it as an area of *partial decalcification*, with obliteration of the dentinal tubules by swelling of the basis substance.   Bennett says that enlarged and thickened tubules can be demonstrated in parts (*loc. cit.* p. 162).
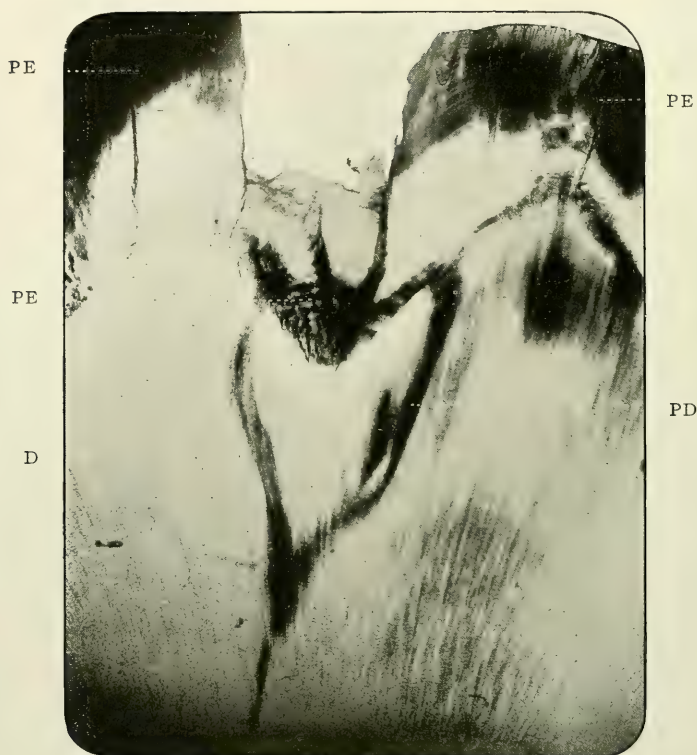
FIG. 134.—An early stage of decalcification of the dentine.   Prepared by grinding.   Unstained.   Magnified 25 times.   p.e. Carious enamel; p.d. Pigmentation area in dentine; d. Normal dentine.

The *Vitalists'* arguments cannot be put entirely out of court because there is no doubt that dentine is endowed, more or less, with a modified form of vitality.   The chief points are these:—

(*A*)   The zone does not form in "dead" teeth.

[1] "Pathologie der Zähne," 1870.
[2] "American System of Dentistry," 1887.
[3] "Caries der Zähne," 1867.
[4] *Trans. Odonto. Soc. of Great Britain,* 1895.

Miller examined both macro- and micro-scopically teeth which had been worn on plates, and he never found one specimen which gave signs of the zone. A large number were subjected to the naked eye test, while about a dozen different "dead" teeth were ground down and examined microscopically.

(*B*) The zone is seen in cases where enamel has undergone attrition and where caries is absent.

(*C*) Narrow bands of translucent dentine sometimes extend even to the pulp chamber. This is never the case with decalcification, which proceeds through the tissues in a fairly regular line.
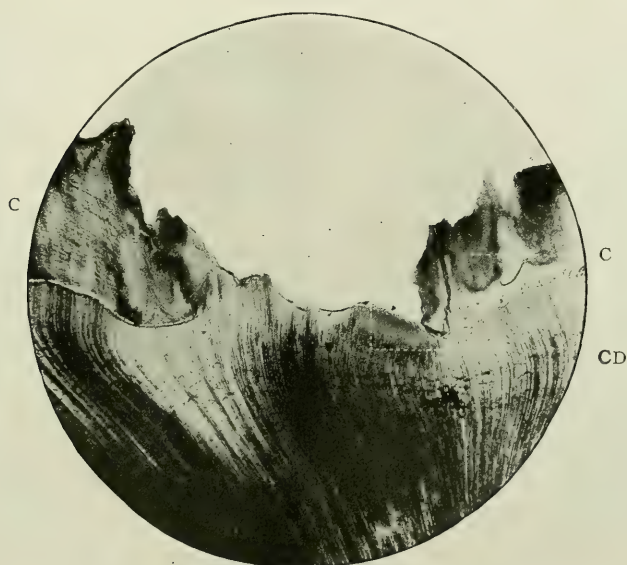
FIG. 135.—A further extension of caries, showing penetration to the cortex of the dentine of the carious cavity. Prepared by grinding. Unstained. Magnified 20 times. c. Carious enamel; c.d. Carious dentine.

(*D*) The chemical analysis of the dentine in the translucent zone does not yield results at all compatible with the decalcification theory. Normal and translucent dentine were tested; the former yielded 72.1 per cent. of lime salts; the latter 71.9 per cent., a difference, as Miller pointed out, quite within the limits of the errors of experiment. Jeserich analysed the translucent and normal dentine of twenty teeth, and his experiments showed that the former contained 69.5 per cent. and the latter 68 per cent. of lime salts. These results do not indicate any decalcification.

(*E*) The zone is difficult to stain with eosin, fuchsine, and other aniline dyes; whereas decalcified dentine is easily coloured by similar re-agents.

(*F*)   The zone cannot be produced *in vitro*.
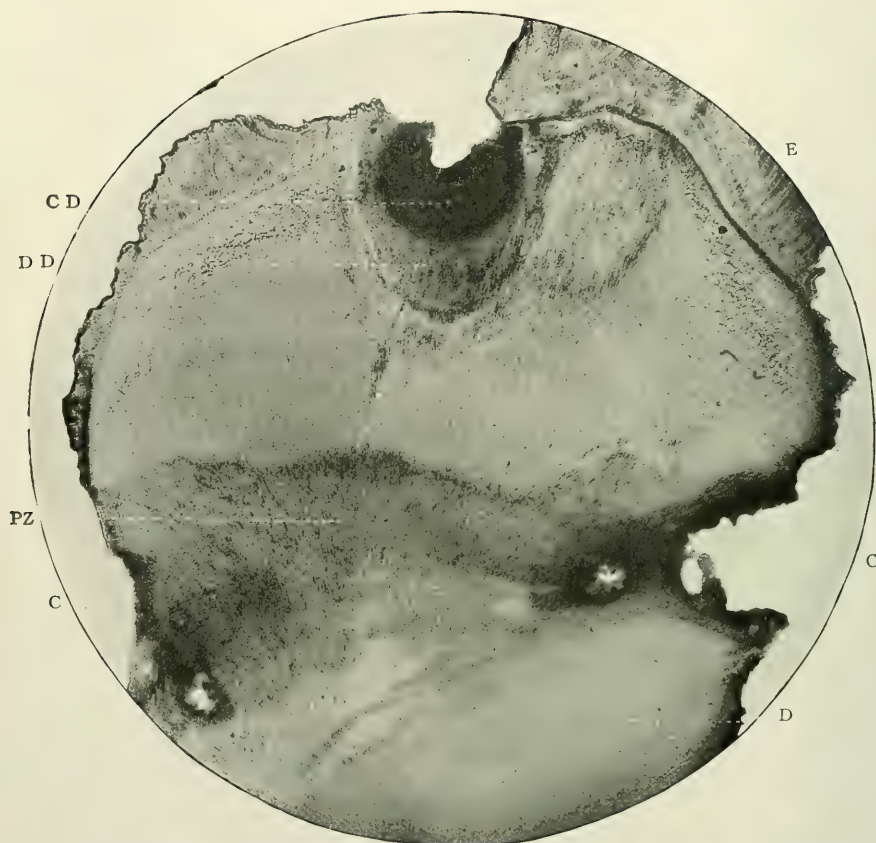
FIG. 136.—A further extension of the carious process in dentine. Most of the enamel was removed during the act of grinding the section. Unstained. Magnified 12 times. E. Enamel; D. Dentine; C. Cavities produced by caries; C.D. Carious dentine, the tubes of which are infected by micro-organisms; D.D. Decalcified dentine; P.Z. Pigmentation zone of decalcified dentine.

In addition, Walkhoff deliberately asserts that there is a diminution in size of the calibre of the tubules, as well as in the diameter of the fibrils.

Wellauer says that there is a contraction of the lumen of the tubule; and Baume, that this contraction leads, in time, to absolute obliteration of the tubules.

On the other hand, the *Devitalists* hold that:—

(*A*) The zone is found in "dead" natural teeth which have been subsequently attacked by caries, after they have been mounted on a plate and worn in the mouth for some time:
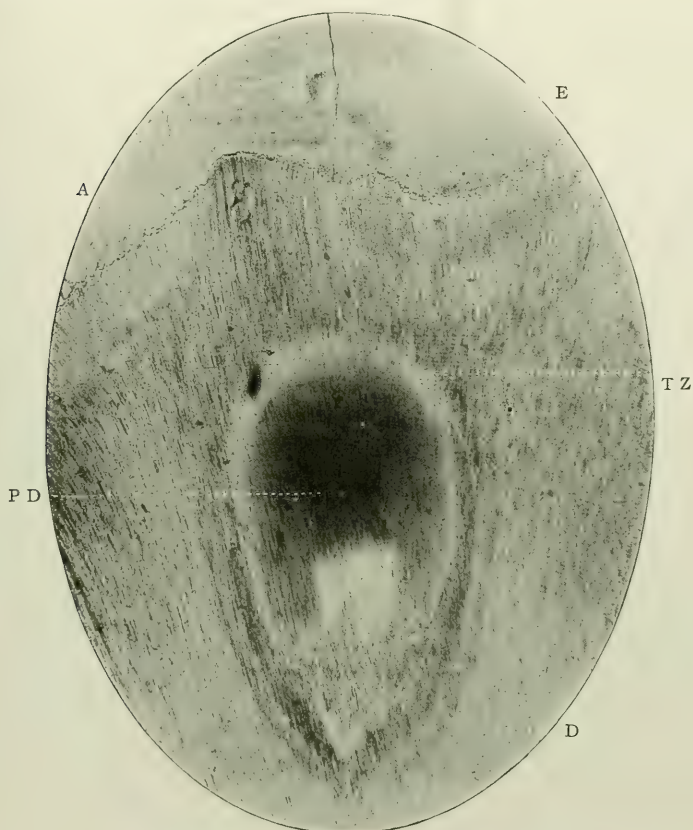
FIG. 137.—Translucent zone in dentine, cut obliquely. Prepared by decalcification in hydrochloric acid, stained with Ehrlich's acid hæmatoxylene. Magnified 35 times. E. Enamel; A. Amelo-dentinal junction; D. Non-carious dentine; T.Z. Translucent zone; P.D. Oblique patch of pigmented decalcified dentine.

(*B*) It exists in cases of partial arrest of caries in teeth, where it might be easily supposed that calcification of the fibrils and tubules was actually taking place:

(*C*) The tubules in the zone are rather enlarged and thickened, presenting "pipe-stem" appearances.

Further, Leber and Rottenstein state that it is softer than the

normal surrounding dentine; and Wedl proved the patency of the
tubules in the zone by immersing in a solution of carmine several
dried sections of the transparent roots of senile teeth. The stain
passed up the tubules quite easily.
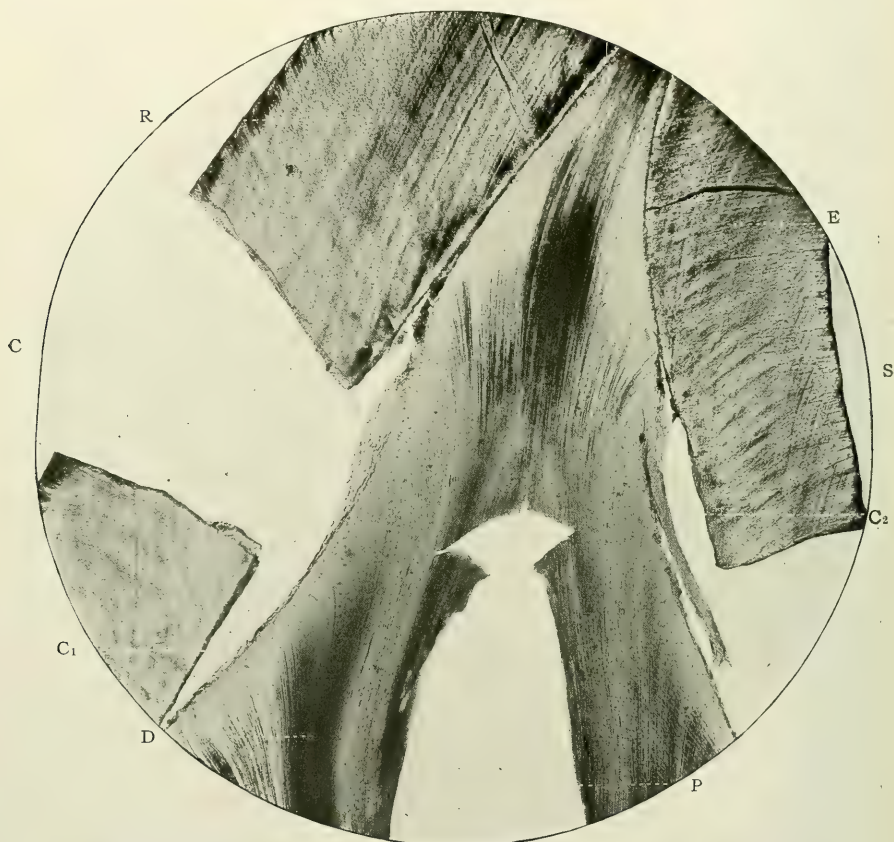
FIG. 138.—Extensive caries of the coronal region of a human canine tooth.
Prepared by grinding. Unstained. Magnified 30 times. E. Enamel; R.
Brown striæ of Retzius; S. Lines of Schreger; D. Dentine; c_1. Carious cavities on
labial aspect of the tooth extending laterally and undermining the enamel;
c_2. The same on the lingual surface; P. Pulp cavity. The enamel has not been
fractured during the act of grinding the section.

The arguments and theories on either side are very cogent,
and one cannot say, in the present state of knowledge, which is
correct. But it is interesting to know that similar appearances have
been found in cases of attrition and erosion of the teeth.
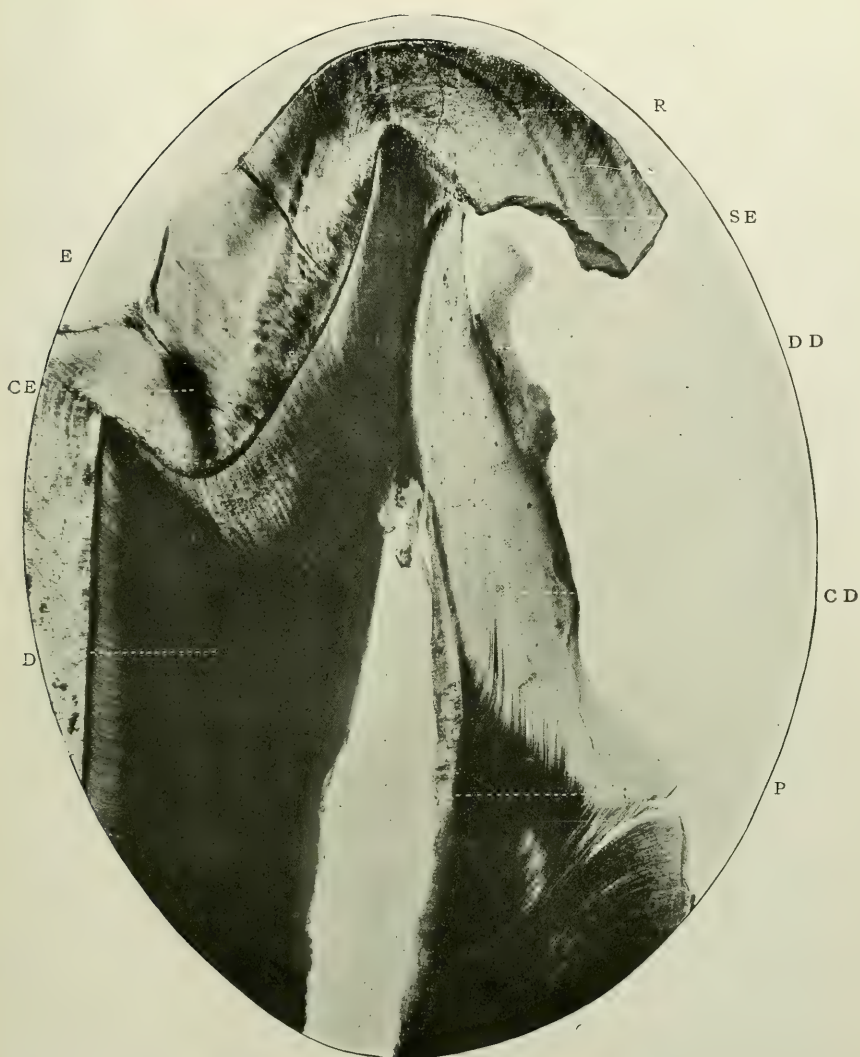
FIG. 139.—Coronal section of a human premolar, with extensive caries of enamel and dentine. The section is rather thick in order to retain the enamel *in situ* without fracture. Prepared by grinding. Unstained. Magnified 15 times. E. Normal enamel; C.E. Carious enamel; R. Brown striæ of Retzius; D. Normal, but very pigmented dentine; P. Pulp cavity; S.E. "Secondary enamel decay;" C.D. Carious dentine; D.D. Disintegrating dentine in cavity on the buccal aspect of the tooth.
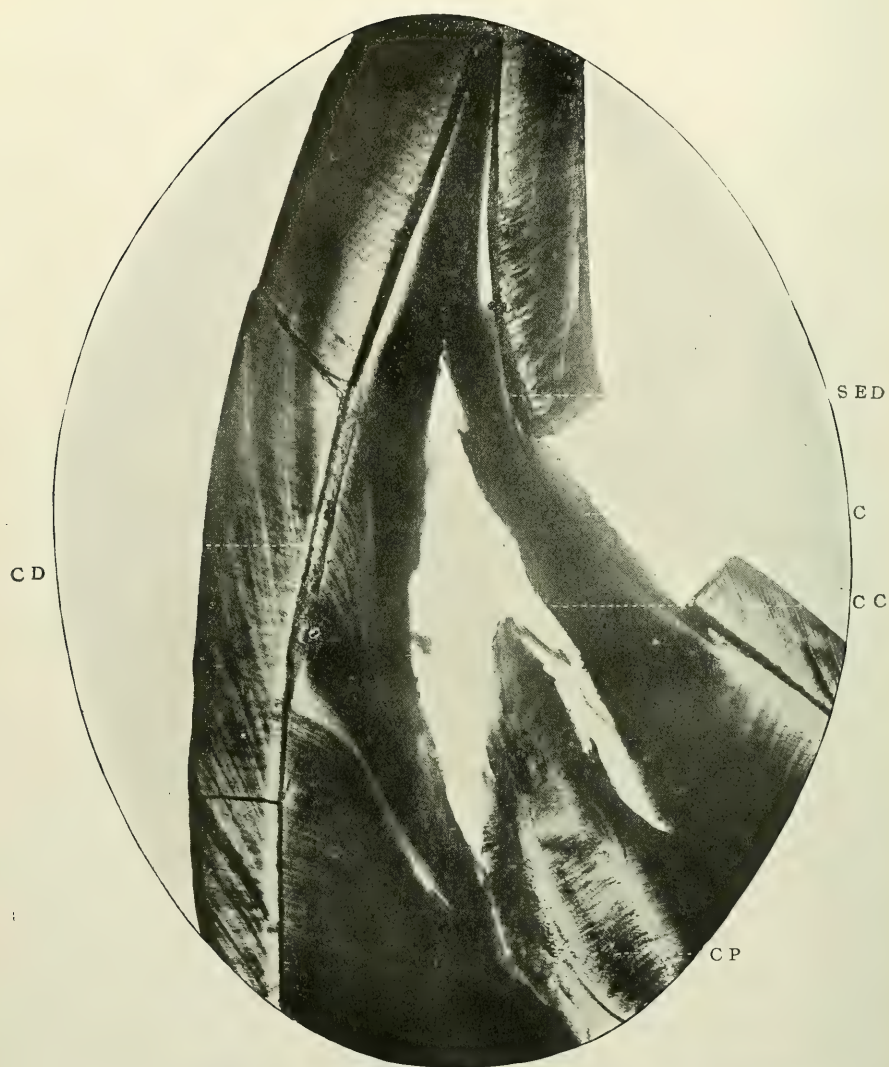
FIG. 140.—Sagittal section of a human incisor tooth. Similarly prepared and magnified as the preceding. C. Carious cavity with deep pigmentation of the dentine, on the lingual aspect of the tooth; C.C. Carious cavity extending towards the mesial and distal sides of the tooth; C.P. Cornu of pulp which has undergone calcification; S.E.D "Secondary enamel decay;" C.D. Caries of the dentine extending in a lat ral direc ion.
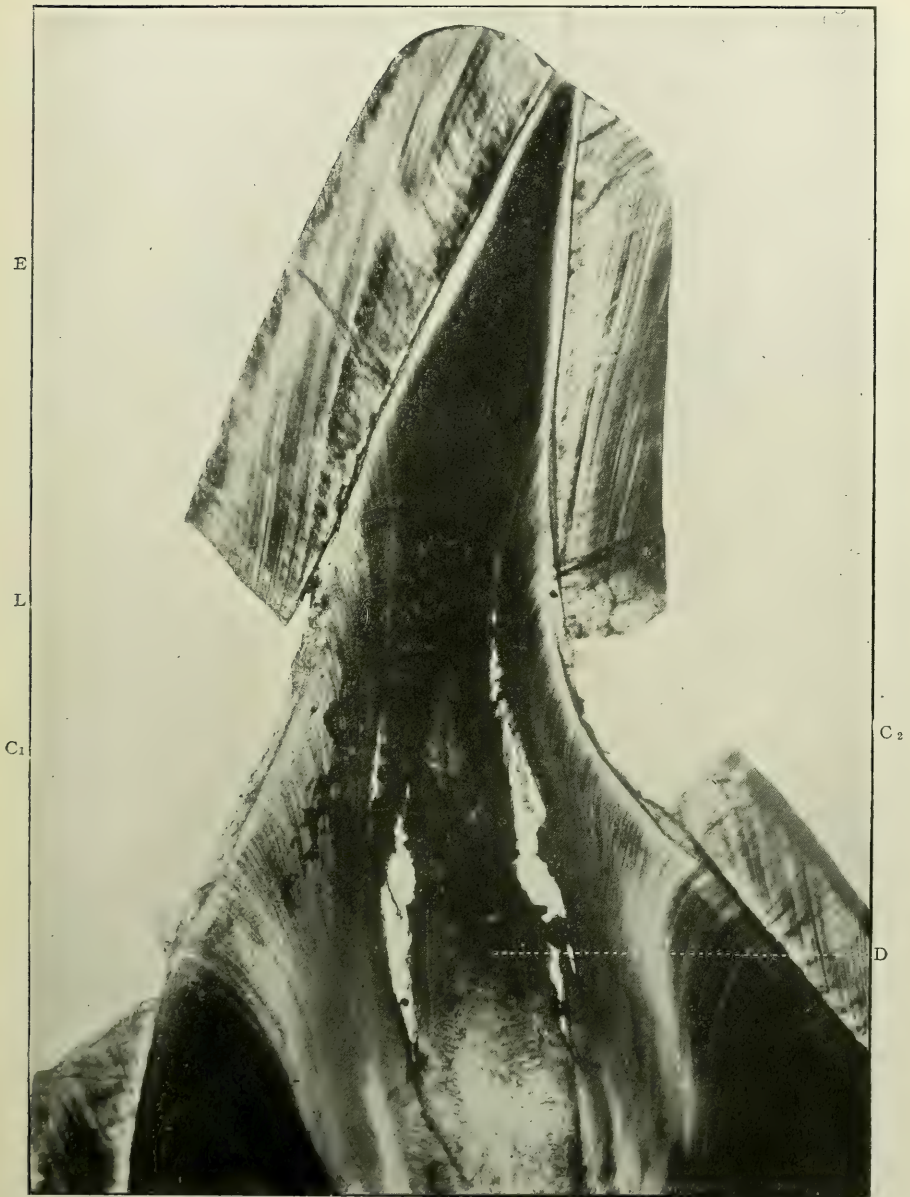
FIG. 141.—Sagittal section of a human incisor tooth. Prepared by grinding. Unstained. Magnified 20 times. E. Normal enamel; c_1. Carious cavity on the labial surface; c_2. The same on the lingual surface; L. Extension of cavity undermining the enamel; D. Carious dentine.
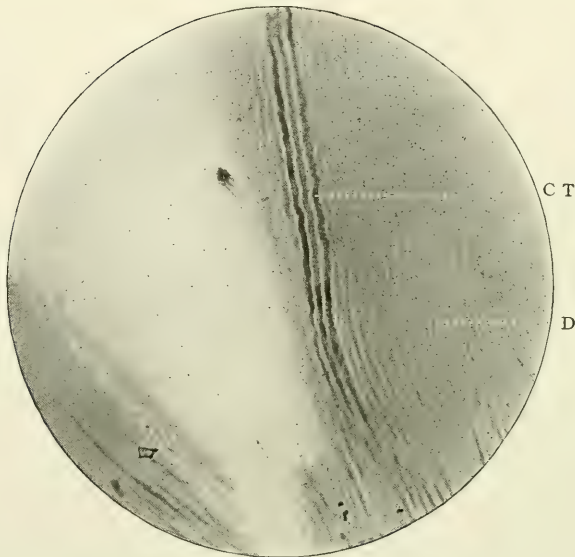
FIG. 142.—Tubular infection of the dentine by micro-organisms. Prepared by the decalcification of carious dentine. Stained by Gram's method. Magnified 250 times. D. Normal dentine; C.T. Micrococci in the tubules
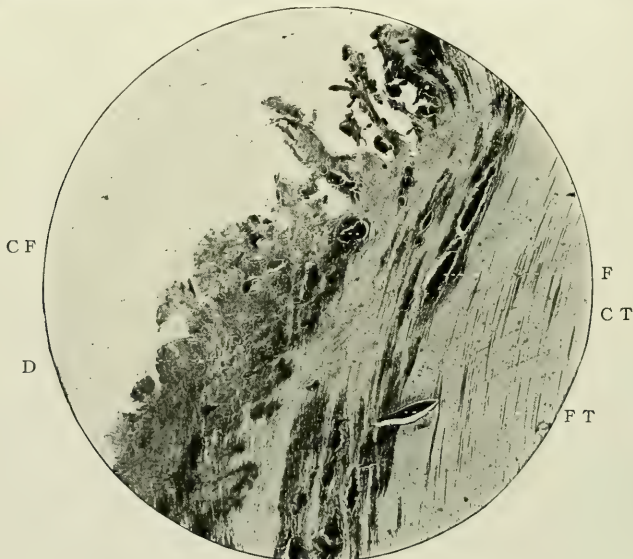
FIG. 143.—Tubular infection of the dentine, with the formation of "liquefaction foci." Prepared as in the preceding. Stained with Ehrlich's acid hæmatoxylene. Magnified 50 times. D. Dentine at margin of cavity, softened and disintegrated by the action of the micro-organisms; F. "Liquefaction focus" running parallel with the tubes; F.T. The same running across the tubes; C.T. Carious infection of the tubules; C.F. Coalescence of several small "foci."

FIG. 144.—Similar to the preceding. Prepared and stained similarly. Lettering similar. D.D. Disruption of the dentine.
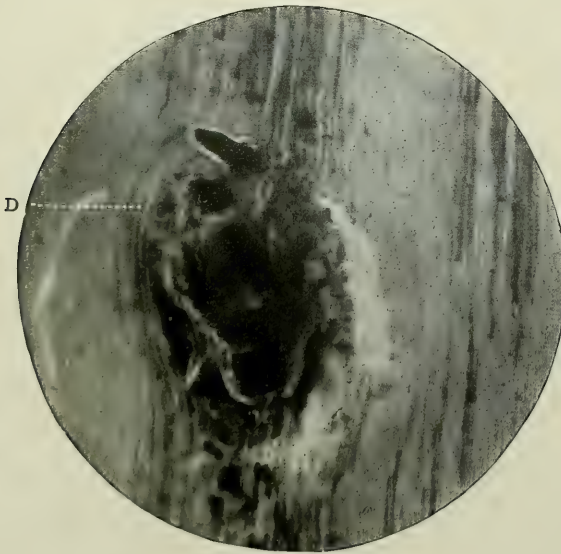
FIG. 145.—A "liquefaction focus" from the preceding figure. Magnified 300 times. D. Deflected tubes, mentioned in the text. See page 135.

Miller observed and described[1] the presence of *opaque* stripes or zones which border on the translucent zone. Under the microscope they appear almost black, and the tubules in these areas are found full of irregular, angular granules or rod-shaped elements. The opaque zones are almost constantly associated with the transparent zones, and usually separate the latter from the normal dentine, or fill up the space between the transparent zone and the pulp.

These differ from the *rod-shaped elements* or *fragments* in the tubules of carious dentine first noticed by Sir John Tomes. They
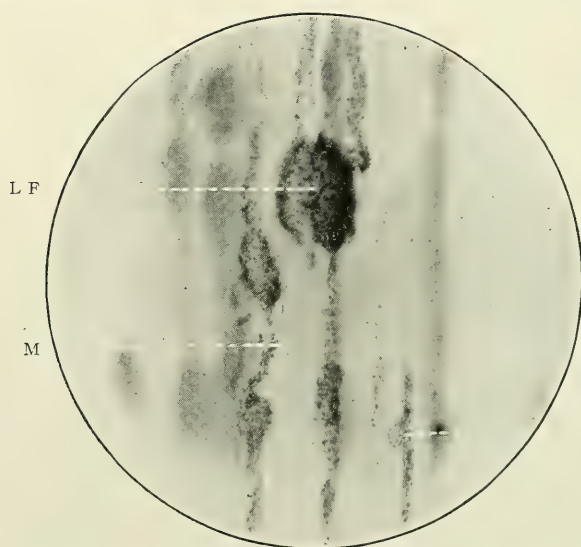
FIG. 146.—Caries of dentine. Magnified 450 times. M. Micro-organisms in dentinal tubes (pure infection); L.F. "Liquefaction focus."

are often seen, and were believed by their discoverer to be "portions of consolidated dentinal fibrils," or "bits of the sheaths of Neumann," or "casts of the enlarged tubules." They are sometimes seen in artificial caries, and if brought into contact with dilute sulphuric acid completely disappear.

Rows of shining irregular granules are also met with in advance of cariesd. It is possible that they have the same origin as the rod-shaped elements just alluded to. Tomes, Magitot, and others described them as "lime-granules," while other observers believe them to have a fatty nature.

[1] "*Trans. Odonto. Soc. of Great Britain*," p. 39, 1895.
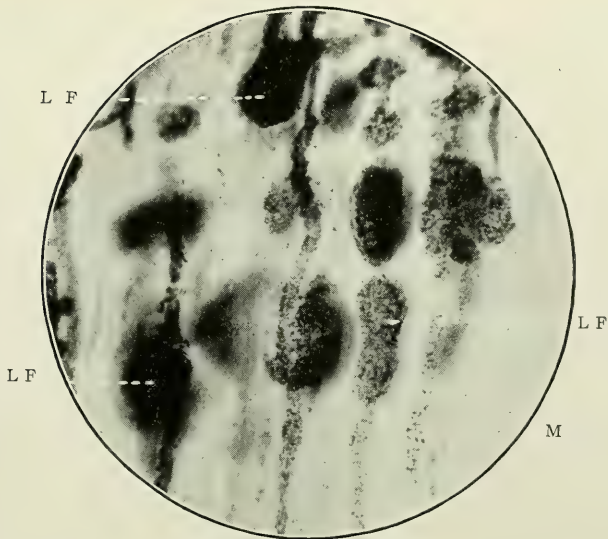
FIG. 14 .—Similar to preceding. Magnified 450 times. M. Micrococci;
L.F. "Liquei ac ion foci."
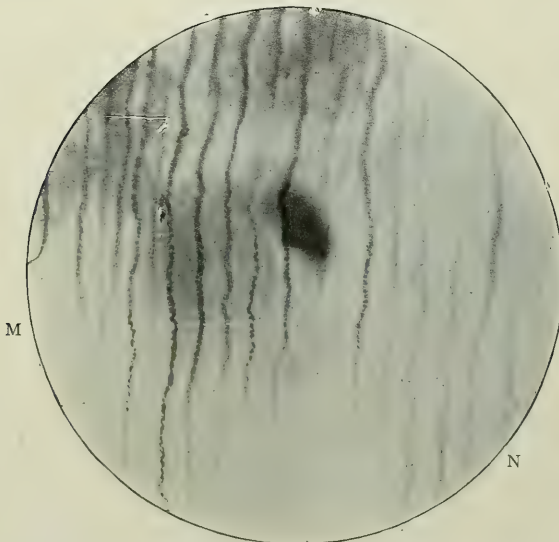
FIG. 148.—Micrococci in the dentinal tubes, from a section of dentine in
which caries was induced experimentally *in vitro*. Prepared by the Author's
method for the production of artificial caries. Stained by Gram's method.
Magnified 750 times. M. Tubule infected by micro-organisms; N. Non-infected
tubule.

9

(v) *Tubular infection by micro-organisms and formation of "liquefaction foci"*

Examined under a low power (16 mm. objective), a section of carious dentine stained by Gram's method, appears of a pale yellow colour, faintly showing the tubules in the unaffected parts, and lines of stain extending inwards from the margins of the cavity. These run in a parallel direction (see Fig. 148), some passing through the whole of the tissue, others traversing part of the distance. In addition, near the margin of the cavity, irregular spaces can be
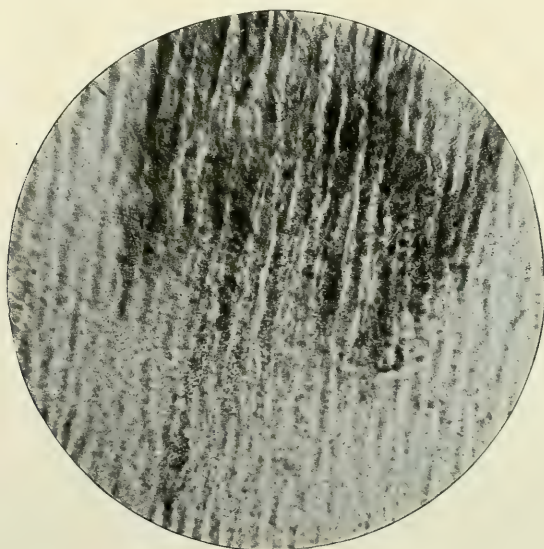
FIG. 149.—Micrococci in the dentinal tubules. Stained by Gram's method. Magnified 550 times.

found, in a line with the stained streaks (Figs. 143 and 144). These spaces vary considerably in shape and size. They are large and irregular near the carious cavity, smaller near the pulp. Here they are less irregular, are oval in shape, and are in direct continuity with the dentinal tubules ("liquefaction foci.")

If now, a higher power be used for examining the section (0.9 mm. homogeneous immersion objective), the stained streaks are seen to be composed of crowds of micrococci and bacilli (see Figs. 148 and 149), and the spaces, in parts, empty, with disorganised tissue at their edges, and in parts filled with micro-organisms. Of all the
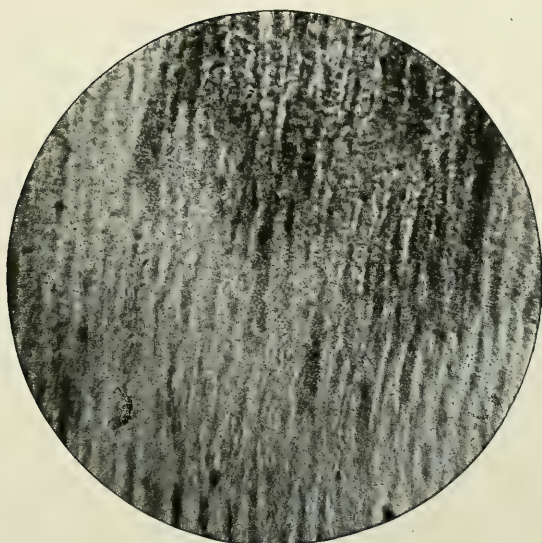
FIG. 150.—Similar to the preceding.
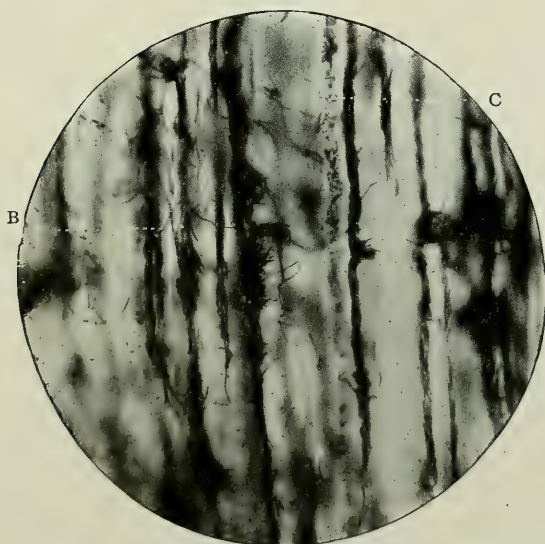
FIG. 151.—Bacilli and micrococci in the dentinal tubules. C. Micrococci; B. Bacilli. (*Photomicrograph by Howard Mummery.*)

varieties, the spherical forms largely predominate, either singly or in pairs, or chains, or clusters. Occasionally the tubes may contain rods, or even threads; a mixed infection also is far from uncommon. (Fig. 151.)

Leptothrix buccalis, and other thread forms, with torulæ, and some micrococci fringe the edges of the carious cavity, as in Fig. 152.

Normal interglobular spaces seem to afford a convenient locality for the housing of the germs. They (the germs) often spread in a lateral direction *viâ* the interglobular spaces; but once having
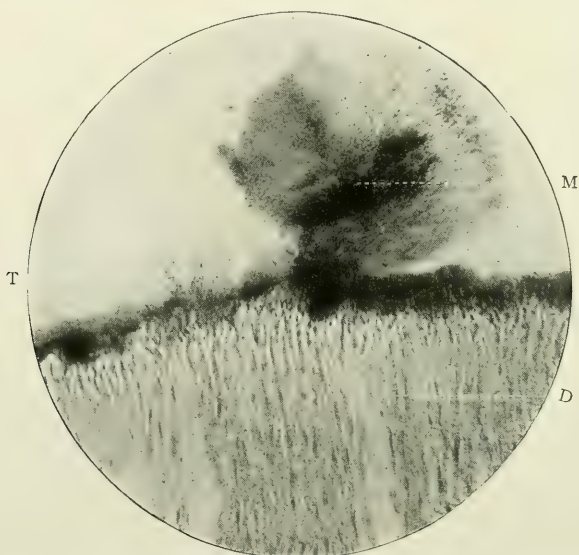
Fig. 152.—Thread forms of bacteria fringing a carious cavity in the dentine. Prepared by decalcification. Stained by Gram's method. Magnified 280 times. D. Carious dentine; M. Mass of micro-organisms; T. A tuft of thread-shaped micro-organisms.

passed them they extend directly inwards towards the pulp, by means of the tubules.

All portions of dentine are not affected; the boundary between the infected and non-infected parts being very regular. This zone is called "the non-infected zone," and shows that decalcification of dentine precedes its infection by bacteria.

After the micro-organisms have once entered the tubules, they easily pass along their lumina. The canals then become choked with micrococci, gradually enlarge, and give way in various places. These masses are called "liquefaction foci," and they correspond
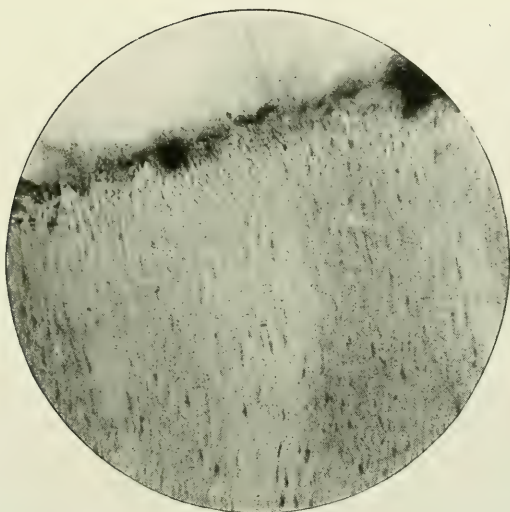
FIG. 153.—Similar to the preceding. Rod-shaped micro-organisms entering the dentinal tubes.
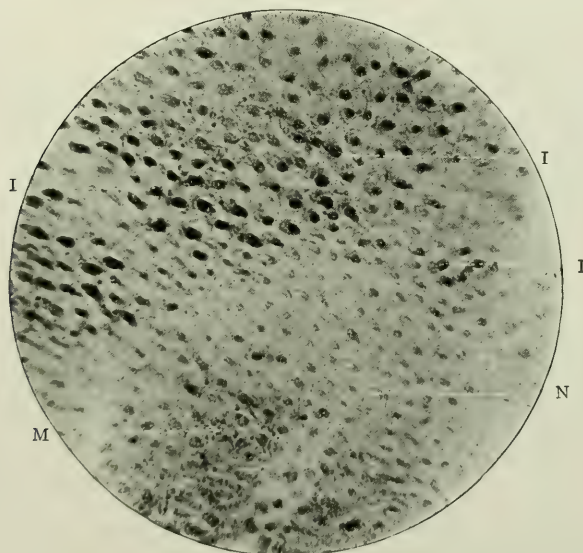
FIG. 154.—The "pipe-stem" appearance of carious dentine. Prepared as in Fig. 152. Stained by Gram's method. Magnified 280 times. I. Infected tubules; N. Non-infected tube; M. Micro-organisms in the matrix of the dentine.

to the "varicosities and globular swellings" mentioned by Sir John Tomes in his earlier writings.

Heider and Wedl ("Atlas zur Pathologie der Zähne," Leipzig, 1869) describe and figure enlargement of the dentinal tubes which possibly are undergoing changes towards those of "liquefaction foci." Isolated from the dentine on the border of the softened carious portion by means of dilute hot hydrochloric acid, they were characterised by a considerable amount of tumefaction, by the
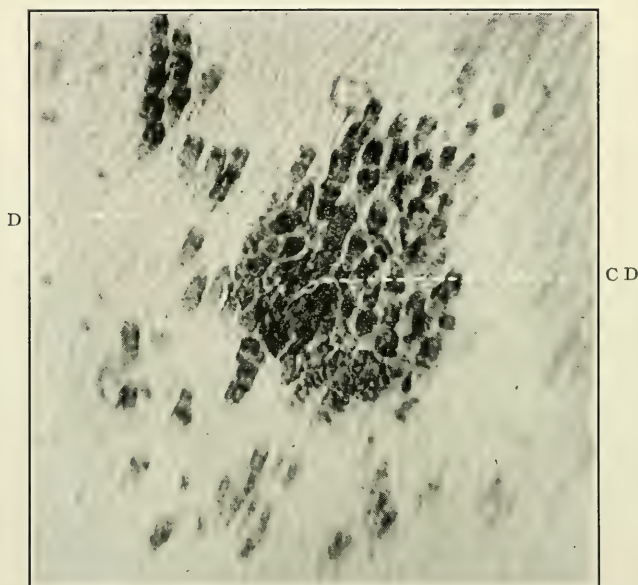
Fig. 155.—Carious dentine; tubes cut obliquely. Magnified 450 times. D. Non-infected dentine; c.d. Carious dentine.

issuing from them of short lateral branches, and by being covered in places with tiny clusters of globules.

If the enlargement of the tubules continues, contiguous "liquefaction foci" become fused, and a space or cavity of an irregular shape is produced. The dentine ultimately becomes more and more cavernous, and so is gradually broken up, and its *débris* finally washed away by the oral fluids.

If transverse sections be made near the healthy dentine, the "pipe-stem" appearance first described by Tomes is visible (Fig. 154). This is the result of the enlargement and thickening of Neumann's sheaths. How this enlargement is brought about is by no

means understood. Miller attributes it, to quote his words, to "the pressure of the fungal masses in the tubules, by which a compression of the walls is caused."

The author believes that the prominence of these tubules is occasioned by two factors:—(i) An actual enlargement of their diameters; and also (ii) An actual but slight thickening of their sheaths.

The expansion is produced by pressure of the micro-organisms in an outward direction on the peptonised or decalcified dentinal
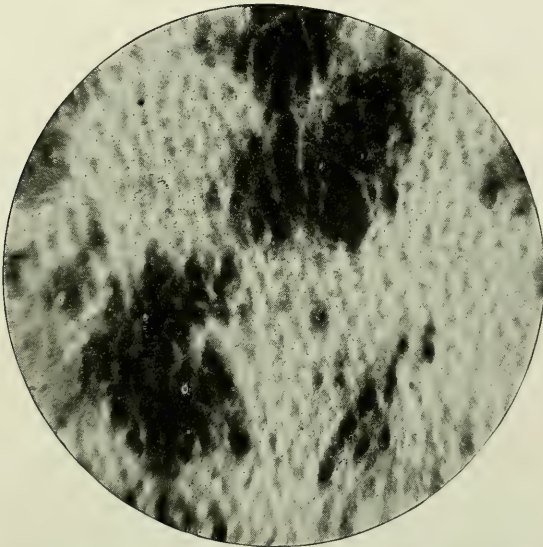
FIG. 156.—Similar to the preceding. Magnified 600 times.

matrix (see Fig. 145). Sometimes the tubules, as at (D), are deflected in their courses by the lateral enlargement of the "liquefaction focus."

The thickening of the specially resistant walls is actually produced by the enzymes of the micro-organisms within, the elastin which they contain, being probably converted into immeasurable quantities of amino-acetic acid.

(vi) *Fusion of Liquefaction Foci and Production of Cavity*

The pressure of the bacterial masses on the softened intervening matrix soon causes the foci to amalgamate. In process of time a
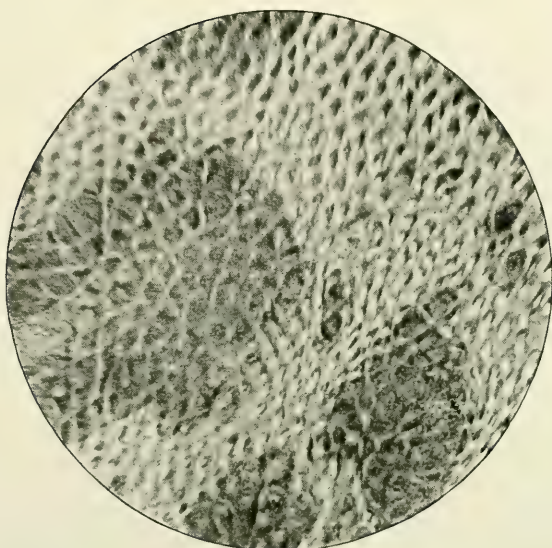
FIG. 157.—Similar to the preceding, but the tubules are cut obliquely
Magnified 600 times.
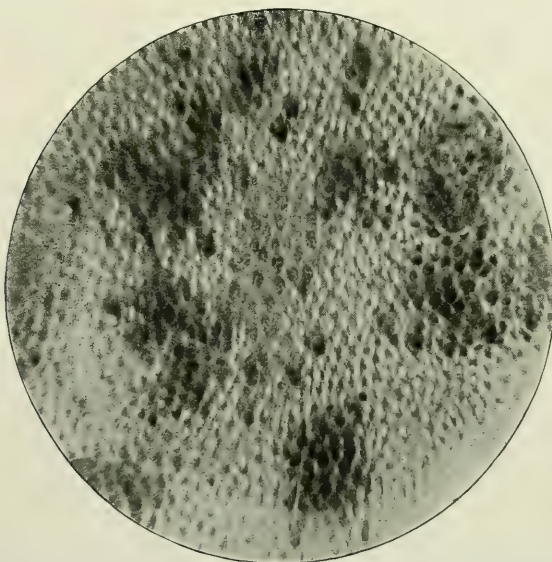
FIG. 158.—Similar to the above, but the tubules are cut slightly obliquely,
Magnified 280 times.

cavity is formed by the coalescence of many foci, its contents being of a soft cheesy consistency, and crowded with micro-organisms. The walls of the cavity become extended by the rapid multiplication and peptonising action of bacteria, and the final stages are reached by the undermining and subsequent fracture of the walls (Fig. 162).

An attempt has been made in the accompanying photomicrographs to illustrate these various stages.
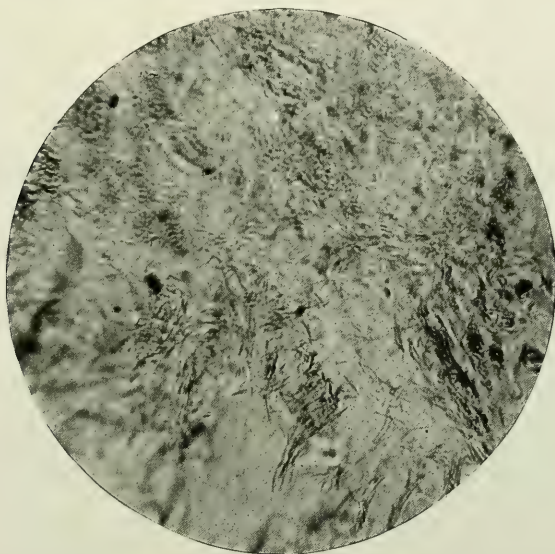
FIG. 159.—Round and rod-shaped bacteria in the matrix of carious dentine. Stained by Gram's method. Magnified 600 times.

(vii) Caries of the Cementum

Caries of the cementum is less common than that of enamel or dentine. It can be well observed on the exposed surface of the palatine roots of maxillary molars when, through septic infection, these have been denuded of their alveolar sockets and periodontal membranes.

The microscopical appearances are interesting, because they show that the micro-organisms gain admission to the short canals occupied by the penetrating fibres of Sharpey, and cause their contents to swell. It proves that these fibres fill these bony channels somewhat similarly to the fibrils in the dentinal tubules. Probably the process of caries in cementum is not quite analogous to that in dentine. In

consequence of its close association with the periodontal membrane, the condition is usually checked in advancing at the border of the tissue. Roots of teeth are often seen in which the dentine has nearly all disappeared, merely a thin shell of cementum being retained *in situ*. The real reason of this would appear to be accounted for by the absence of tubes or lacunæ in the homogeneous layer of the dentine the non-tubularity thus prohibiting here the admission of micro-organisms.

(viii) *"Arrested" Caries*

Little is known of the physical, chemical, and histo-pathological changes which bring about this condition. In the enamel, pigmen-
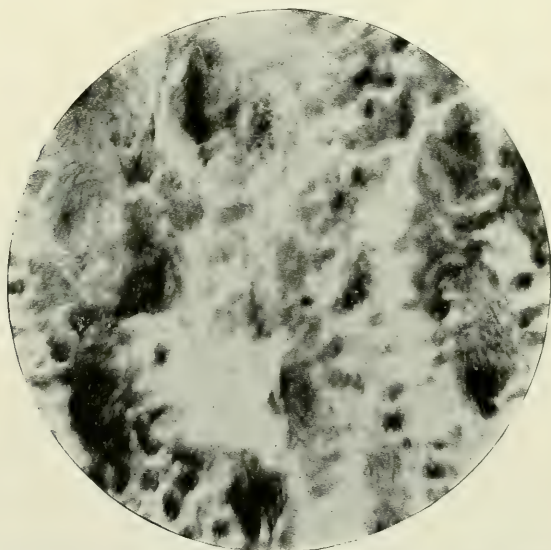
Fig. 160.—Masses of micro-organisms in the matrix of very carious dentine. Staining and magnification as in the preceding figure.

tation extending inwards to varying depths is noticed; the intercolumnar substance is well-defined; the rods themselves present a homogeneous appearance. The brown striæ of Retzius and Schreger's lines may or may not be entirely obliterated (see Fig. 200). In the dentine, the circumferential tubes are occluded, the refractive indices of tubes and matrix thus approximating. In the pulp, adventitious dentines of one or more types may generally be found.

(ix) *The Micro-organisms of Dental Caries.*

The micro-organisms normally found in the human oral cavity are of all kinds: cocci occur most abundantly, but also bacilli thread-forms, sarcinæ and spirilla. It is probable that, of the first named the *Streptococcus viridans*, the *Staphylococcus pyogenes aureus* and the *Diplococcus pneumoniæ* are the commonest of the pathogenic varieties. In addition many aërobic and anaërobic sporeforming bacilli are taken in through the media of air, food, and water (see Chapter XVI).
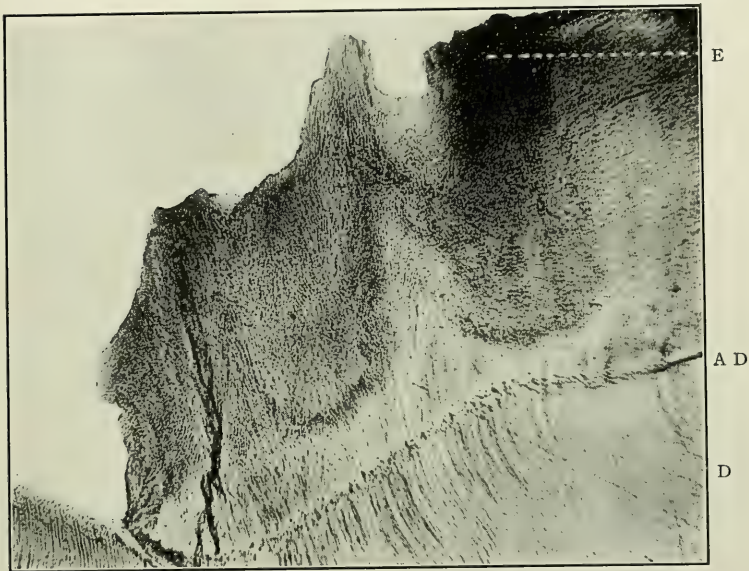
FIG. 161.—Enamel in "arrested caries." Magnified 90 times. E. Carious enamel; A.D. Amelo-dentinal junction; D. Normal dentine.

It is difficult to determine with any degree of accuracy the morphological and biological characteristics of the actual producers of dental caries; it is so extremely easy, even with the utmost care, to obtain bacteria for experimental purposes that are incapable of causing the initial decalcification of the enamel. Nor is it yet ascertained whether the bacterial factors which may occasion the dissolution of the peripheral hard parts of the teeth are the same as those inhabiting the dentinal tubules, or the surfaces of carious cavities. The saliva carries with it, and sweeps into such cavities, adventitious bacteria. Others from the oral mucous membrane, the

gingival troughs, the alveolar "pockets," the tonsils and pharynx, add their quota to the varieties of the oral flora and make real discrimination between the caries-producing and the non-caries-producing organisms very difficult. On microscopical examination of a fragment of carious enamel or dentine, multitudes of bacteria are observed: but it is almost impossible to affirm with certainty whether those present are such-and-such organisms. Identification is often impossible. The bacteria of caries as Goadby[1] points out appear to be influenced very largely by their environment; and it is quite possible that the actual producers of the dissolution of the enamel are often overwhelmed by the preponderating growth of others, which, as secondary agents of "dental decay," penetrate the dentinal tubules and peptonise their walls.

Several facts, however, would seem to stand out pre-eminently. The bacteria in superficial parts are aerobic; those beneath, in the vanguard of the advancing host, anaerobic or facultative aerobic; many as yet, cannot be cultivated on the ordinary laboratory media; the superficially placed bacteria produce proteolytic enzymes, others fermentation with acid production; no liquefaction of undecalcified dentine takes place by means of the proteolytic enzymes; and finally it is practically impossible to differentiate between pathogenic and non-pathogenic forms. For according to Emery ("Immunity and Specific Therapy," 1909), "any bacterium will produce disease if it grows in the tissues of the living body, and all bacteria—those growing only at very high or very low temperatures or on media very poor in nitrogen perhaps excepted—will do so if the necessary degree and form of immunity is not present."

As far as is at present ascertained, it may be briefly stated that the micro-organisms found in the superficial parts of carious dentine are either liquefiers of that tissue, when it has already been decalcified to a certain extent, or are merely enzyme producers. To the former group belong, in alphabetical order, the *Bacillus furvus*, *Bacillus gingivæ pyogenes*, *Bacillus liquefaciens fluorescens motilis*, *Bacillus mesentericus fuscus*, *ruber*, and *vulgatus*, *Bacillus plexiformis*, *Bacillus subtilis*, and *Proteus vulgaris;* to the latter the *Sarcina alba*, *aurantiaca* and *lutea*, *Staphylococcus albus* and *aureus*, and *Streptococcus viridans*. Those isolated from the deeper layers of carious dentine are the *Bacillus necrodentalis*, and *Staphylococcus albus*. A third class includes the chromogenic bacteria. Fringing the edges of carious surfaces are many thread-like organisms. These often in-

[1] "The Mycology of the Mouth," 1903.

clude *Leptothrix innominata, Leptothrix buccalis maxima, Streptothrix buccalis,* and the curved and spiral forms described as the *Spirillum sputugenum,* and *Spirochæte dentium.*

EPITOME OF MICROSCOPICAL APPEARANCES OF CARIES

1. *Nasmyth's Membrane* shows mere pigmentation.
2. *Enamel.*—The rods are separated; their striæ pronounced;

FIG. 162.—Final stages in the destruction of carious dentine. Prepared by decalcification. Unstained. Magnified 40 times. D. Carious dentine; P, Pulp cavity.

are granular, and broken down. Bacteria pass between them and take their place in "secondary enamel decay." Three zones are present, viz., those of partial decalcification, complete decalcification, and "secondary enamel decay."

3. *Dentine.*—Tubules filled with micrococci and bacteria; "lique-faction foci" are formed. "Tobacco-pipe appearances" and "Zone of translucency" are noticed. The opaque zones of Miller frequently exist. In some places rod-shaped fragments fill the tubules. Homogeneous layer usually non-infected.

4. *Cementum.*—Fibres of Sharpey are attacked by micro-organisms, the dentinal surface meanwhile unaffected.

CHAPTER V

THE DISEASES OF THE DENTAL PULP

MICROSCOPICAL ELEMENTS IN:—(i) Hyperæmia; (ii) Acute and Chronic Inflammation; (iii) Infective Gangrene of the Pulp; (iv) The Pathogeny of Gangrene of the Pulp.

GENERAL CHARACTERISTICS

In nearly every case of dental caries there are certain morbid processes found in connection with the dental pulp. The majority of the pathological conditions met with in this organ are due to causes operating from the outside, only a few being determined by and dependent on endogenetic disturbances.

Ætiology.—The causes of diseases of the dental pulp generally may be classified as (*A*) Predisposing, (*B*) Exciting.

(*A*) *Predisposing Causes.*—(1) *General:* (*a*) Physiological, *e.g.:* Heredity, age, sex; (*b*) Pathological, *e.g.:* Marasmus, long-continued fevers, gout and similar dyscrasia; (2) *Local:* Anatomical peculiarities of the dental tissues.

(*B*) *Exciting Causes.*—(1) *General:* Effects of disease of (*a*) Vascular system, *e.g.:* Anemia, chlorosis, gout, etc., and (*b*) Nervous system, *e.g.:* Neurasthenia, etc.; (2) *Local:* (*a*) Apparent—Effects of dental caries; (*b*) Non-apparent—Thermal, chemical, and electrical stimulations, etc.

It is extremely probable that morbid affections of Nasmyth's membrane or enamel *per se* (both products, as already described in Chapters II and XII, Vol. I, of the stomodæal ectoderm) exert some unknown influence on the subjacent tissues. This, and the two following Chapters, however, are concerned with the variations that take place in the pulp at periods of time antecedent and posterior to the carious penetration of its containing cavity: as also the histological elements in the pathogenic changes it undergoes through traumatic and degenerative lesions.

It is necessary, at the outset, to again remind the student of the fact that the pulp has no pathology peculiar to itself. It shares with other organs of a highly vascular nature phenomena of an

identical kind, differing mainly in the particulars that it is enclosed in a bony casing which prevents much inflammatory exudation, or swelling. This confinement in hard unyielding walls probably prevents, in no slight degree, the tendency for an exposed pulp to cicatrize, and induces, in the majority of cases of acute inflammation, the death of the pulp in part or in whole.

Peripheral carious stimulation of the dentine is accompanied by destructive as well as constructive metamorphoses; tissue waste and tissue repair go on side by side. At first the soft parts alone suffer; the dentinal fibrils and their enclosing tubules, in parts of their courses, are affected and soon become disorganised, the blood-vessels and tissues of the pulp meanwhile undergoing hyperæmic and other changes. Thus a superficial carious patch beneath the cortex of the enamel is associated with marked cellular activity on the part of the pulp; while there is a loss of substance externally, there is a gain internally. This is exemplified in the formation in certain circumstances of "dentine of repair." In other words, caries, even in its early stages, usually leads to a deposit of new adventitious dentine on the surface of the pulp.

But, later on, bacterial agencies multiply and accumulate; advancement renders them still more potent; and development means destruction. For now not only do the dentinal tubules and matrix also become involved in the general dissolution, but any adventitious tissues that may have been developed, rapidly break down, and soon the work of demolition is complete. A study of these phenomena possesses many points of profound interest.

Hyperæmia of the Pulp

This condition is analogous in all respects with active or arterial hyperæmia and passive or venous hyperæmia occurring in other soft tissues of the body. It may be partial or complete—confined to one of the cornua or the coronal, cervical, or radicular portions of the pulp cavity, or distributed throughout the whole of the soft tissue. It is probably dependent, in the first instance, on irritation of the dentinal fibrils, produced by various causes, and may pass quickly on to acute hyperæmia and inflammation of the pulp. It has been called "Irritation of the pulp," an incorrect appellation, for irritation is the cause, not the actual disease. The first change from normal to an abnormal state of the pulp is not irritation but hyperæmia—if slight it is usually *regional*, if intense *diffuse*.

Etiology.—Any factor that determines an increase of the flow of blood in the tissue is the cause of hyperæmia. Thus it may be ascribed to:—(i) Caries, (ii) Injury to the dentine, which sets up dentinal irritation, (iii) Early stages of cold, (iv) Rheumatic affections of the jaws, and (v) possibly Hydrargyrism.

Secondary Changes.—Resolution or inflammation, most commonly the latter.

GENERAL CONSIDERATIONS

The nutritional well-being of the pulp depends upon the proper regulation of its blood supply, which is governed by the influence of the sympathetic nerves of the unstriated muscle fibres in the walls of the arteries, the elastic tissue in the *tunica media* of the vessels maintaining simultaneously the necessary "tone." When the usual amount of blood exceeds this physiological limitation, pathological hyperæmia is induced and may be (A) Active or arterial, or (B) Passive or venous, according to which set of vessels are most chiefly affected.

(A) Arterial hyperæmia may be induced by any condition which either (i) paralyzes the vaso-constrictor nerves or (ii) stimulates the vaso-dilator nerves, or (iii) weakens the *tunica media* or (iv) removes the extra-vascular pressure. All these factors may act singly or in concert with one another.

If a sufficiently large area of dentine has been exposed during the course of dental caries, various forms of irritation may set up a localized regional hyperæmia. Such forms of irritation are the chemical or bio-chemical products of decomposition of liquid or soft food, vitiated oral secretions, thermal changes in the mouth, and drugs medicinally or artificially applied for therapeutic purposes. Cold paralyzes the vaso-constrictor nerves; heat, the vaso-dilators, while the alteration of the protoplasmic contents of the dentinal tubes by the products of bacteria and the use of chemical reagents probably weakens the vessel walls.

(B) Passive or venous hyperæmia occurs much more frequently than arterial hyperæmia. It is due to an abnormal obstruction to the outflow of blood from the veins of the pulp, being dependent upon local conditions. The obstruction is generally at the apical portion of the roots of the teeth, where the mechanical unyielding of the hard dentinal walls combined with the absence of a collateral circulation causes its development to the fullest degree.

The microscopical changes in the pulp affected by venous conges-

10

tion, which will be presently fully described, may be summarized as follows:—A capillary and venous dilatation is followed by the organ becoming deeply reddened; the axial and peripheral blood streams in the veins become confused; the erythrocytes are densely crowded together; and stasis (the cessation of flow of the blood current) and emigration of the colourless blood cells supervene. The dilatation of the capillaries and veins arises from the loss of balance of the hæmodynamic pressure caused by the lessened resistance of the blood stream to friction, through its slowing down. A transudation of *liquor sanguinis* through the endothelial walls is favoured and accelerated by the intra-vascular pressure; and more or less œdematous conditions quickly ensue, because of the absence of lymphatic vessels from the pulp. If these conditions remain unrelieved, acute inflammation takes place.

SPECIAL HISTOLOGY

Viewed from a clinical aspect, it may be stated that the commencement of caries is marked, as a rule, by one of two distinct types of lesions: (1) the not uncommon clean-cut cavity, which by its general appearance suggests erosion of the enamel and dentine; and (2) the usual cavity of "decay." The former is distinguished by its position on the cervical portion of the labial aspect of the anterior teeth, and by its intense hyperæsthesia on receiving interrupted tactile impressions; the latter is recognised by its inability to transmit slight functional impulses to the pulp. Microscopically the difference between these two classes is well defined, the first-named particularly presenting marked deviations from the usual type.

(1) The sub-enamel region of the dentine contains not only the usual dentinal tubes but also areas occupied by large interglobular spaces, which are distributed with more or less regularity throughout its substance. Micro-organisms are present in enormous numbers at the margin of the cavity, and fill the tubules for varying distances. Opposite the breach of surface a corresponding deposit of adventitious dentine with enlarged irregular tubules is observed. The dentogenetic zone, that layer of tissue "on the borderland of calcification" is increased in thickness, and exhibits a greater quantity of calco-globular masses than normally. They are, however, very small. In the pulp, slight hyperæmia and cell proliferation have certainly occurred in this locality and its neighbourhood; and the peripheral cells, which present many of the appearances

of the so-called odontoblasts, are multiplied greatly. Beyond, the tissues may be considered to be normal, with the exception perhaps of the smaller blood-vessels, whose lumina are more or less increased in size. Rounded cylindrical deposits of newly formed but uncalcified dentine constantly exist in the central portions of the

FIG. 163.—Hyperæmia of the pulp *in situ*. Prepared by the Author's process. Stained with Ehrlich's acid hæmatoxylene. Magnified 50 times. P. Pulp tissue; I.T. Inflammatory products; B. Enlarged blood-vessels; O. Odontoblasts D. Dentine.

pulp, and point to a degenerative process. The changes from the normal to the pathological areas are very gradual, no sharp line of demarcation cutting them off from the other parts of the soft tissues.

Referring to the statement just enunciated that "the so-called

odontoblasts are multiplied greatly," it must not be inferred that these cells are merely numerically increased. They are profoundly modified, inasmuch as they now possess certain new characteristics. Their nuclei have become elongated and flattened, and are rendered very prominent when any of the nuclear stains have been used, and perhaps they are more granular than usual. The cell walls are indistinguishable, chiefly from the fact that each odontoblast is compressed laterally by its neighbours. In some instances they are gathered into sheaves, as in fibroid degeneration or atrophy of the pulp. Some observers might describe the appearances as being due to an indirect splitting-up of the cells: and it is not difficult to conceive of an odontoblast, when once fully formed, undergoing mitotic changes. In sections prepared by Weil's method, microscopically the tubules in the primary dentine below the breach of surface are unaffected by stains, and clearly differentiated from other tubules, having been, perhaps, fully calcified throughout their extent, and a band of altered pulp tissue may extend right across that organ.

Thus, at the very outset, two most remarkable conditions attract attention. These are the multiplication of the numbers of interglobular spaces, and also cytogeny of the so-called odontoblast cells—phenomena which are entirely absent from all ordinary conditions.

There would, therefore, seem to be some connection between the subjective symptoms of pain and these fresh developments—or at all events one of these fresh developments—and this leads one to the conclusion that the degree of the sensitiveness of these cavities is dependent partly on the increase or diminution in the numbers of the interglobular spaces, and partly on the anatomical relationships which hitherto existed between the hard tissues. The author believes that the sensitiveness of these marginal cavities is due, in a great measure, to the actual exposure of the terminations of the dentinal fibrils, which is associated with that developmental error where the edge of the enamel and cementum do not meet, as they normally do *bout à bout*.

Bödecker,[1] in speaking of dentinal irritation in the case of ordinary caries, attributes the sensations of pain to "alternate contractions and expansions of living matter" in dentine and enamel, "conveyed from the periphery to the centre of the tooth, these intense contractions being induced by highly irritating agencies."

But it must be remembered that it is only mechanical stimulation, at first, of the floor of these particular cavities that gives rise to pain;

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[1] "Anatomy and Pathology of the Teeth," p. 288, 1894.

and it must be inferred with greater accuracy in the light of present knowledge of the physiological stimulation of nerves and protoplasm generally, that the pathic disturbances are due here to direct impulses, which pass by means of the dentinal fibrils from the protoplasmic contents of the interglobular spaces to the ultimate telodendria of the sensory pulp neurones.

The occurrence of additions to the numbers of the long odontoblast cells does not admit of quite so easy an explanation. The writer[1] just quoted has indirectly noticed, although he has not figured
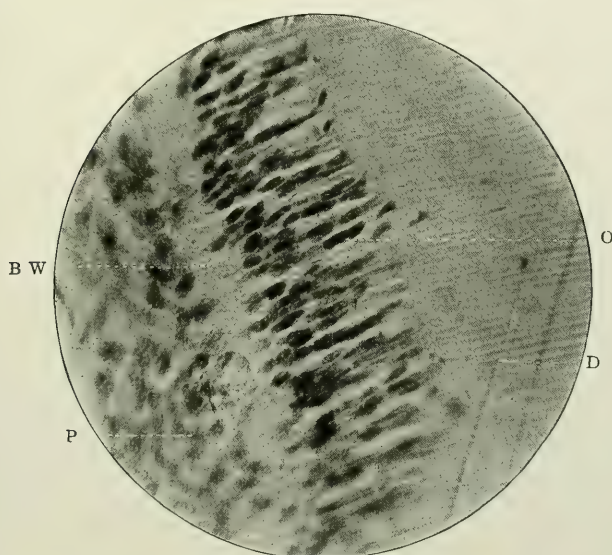
FIG. 164.—Odontoblasts in acute inflammation of the pulp. Prepared similarly to the preceding. Magnified 230 times. o. Odontoblasts; D. Dentine; B.W. Basal layer of Weil; P. Pulp tissue.

this phenomenon. He says:—"The first change in the affected pulp-tissue is its reduction to an embryonal or protoplasmic state" —a statement which is certainly not verified on examination of properly stained microscopical preparations. Further, he proceeds:— "Should the lymph-tissue be reduced to its embryonal conditions as above indicated, the protoplasm present before transformation into basis-substance reappears, and *may break up into odontoblasts* or into *osteoblasts*. In the former case, the result of irritation of the pulp-tissue will be *dentine*, in the latter *bone*." The method of thus

[1] *Op. cit.*

interpreting the genesis of these lime-bearing cells is crude and illogical.

The local increase in the numbers of the odontoblasts may demonstrate that, in certain situations, there is a greater need for the higher and more sustained exercise of their functions, these functions, probably, consisting mainly of shielding the delicate pulp from incoming dangers; not by the production of dentine matrix, but by physiologically creating a larger or more concentrated area of trophic influence or control—if one may so speak—on the surface of that organ, whereby its vitality may be retained until the latest possible
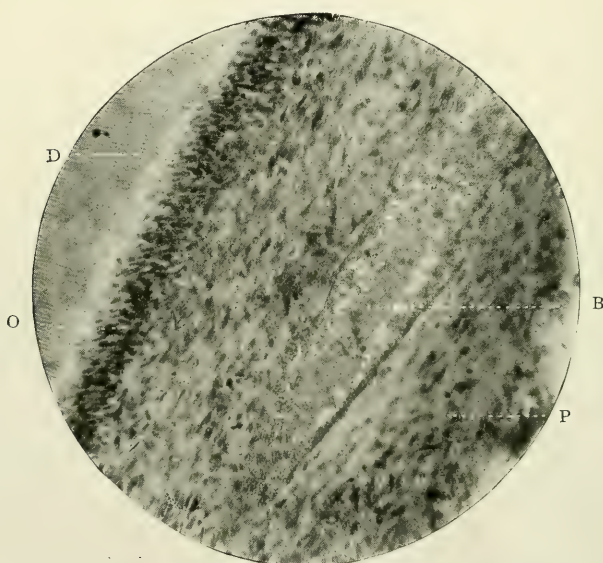
FIG. 165.—Acute inflammation of the pulp *in situ.* Prepared as in the preceding. Magnified 130 times. D. Dentine; o. Inflamed odontoblasts; P. Inflamed pulp tissue; B. Small vein with axial current of blood corpuscles and, at the sides, emargination of the leucocytes.

moment; or it may be that the odontoblasts have merely undergone mitosis. This is probably the correct view to hold.

True formations of compact bone are most rarely found in the tooth-pulp; several cases, however, have been recorded (see pp. 68, 69), but calcareous deposits are exceedingly common.

Compact osseous tissue consists of Haversian canals, concentric and intermediary lamellæ, lacunæ, and canaliculi, with blood-vessels, osteoblasts, connective tissue, branched bone corpuscles, and minute lymphatic systems. (See Chap. VI, Vol. I.) And if these com-

ponent parts are non-existent, it is a mistake to pronounce the new formation bone.

(2) Turning, in the second place, to cases in which the dentine is well developed and free from irregularities, the subjective pain symptoms do not, as a rule, appear until there is almost penetration into the pulp chamber, no matter how rapidly the carious encroachments may take place. But the pulp exhibits similar microscopical characteristics to those already detailed, the most obvious being cell-proliferation and odontoblast multiplication with isolated cylindrical calcoglobulin formations in the neighbourhood of the vessels. Regional hyperæmia is often present—that is, the capillaries, arteries, and veins are rather larger, have thinner walls, and are more tortuous than normal.

Acute Inflammation

Definition.—This is one of the terminations of hyperæmia, and also one of the results of dental caries. It is one of the commonest affections of the pulp, because of the anatomical peculiarities of the parts, and of the prevalency and numbers of its causes. *Synonym:* Pulpitis, a hybrid term.

Etiology.—(i) Caries; (ii) The incautious use of drugs; (iii) Fillings of an irritant or thermal conducting nature; (iv) Traumatism; (v) As an extension of inflammation from the alveolo-dental periosteum. The latter is septic in origin and occurs in teeth whose roots are more or less incomplete, and whose apical foramina are still very patent.

Terminations.—Resolution, organisation, suppuration, or gangrene.

Signs and Symptoms.—The Galenic signs of acute inflammation are manifested in the pulp as elsewhere, but vary greatly in intensity. Thus pain (*dolor*) is the greatest, whilst swelling and heat (*tumor et calor*) owing to its circumscribed environment are the least. The pulp macroscopically becomes very red (*rubor*). The first is due to pressure upon the nerve bundles and the great tension produced by the hyperæmia; and the others are due to hyperæmia, leucocytic emigration, serous exudation, proliferation of "fixed" tissue elements, and the relatively large amount of blood in the parts.

GENERAL HISTOLOGY

Inflammation is the complete local reaction of the tissues to injuries and lesions of various kinds.

"In recent years it has become more and more evident that the only theory which allows the full meaning of inflammation to be grasped, is the broad biological conception which recognises in inflammation an adaptive protective, and reparative tendency common to the reactions to injury among all animals." (Hektoen and Riesman. "*A Text-book of Pathology*," 1901.)

To Cohnheim[1] and Metchinkoff[2] belong the credit for a great deal of the early and late knowledge of this subject. "Inflammation brings into operation a number of factors to counteract harmful agents, protect the organism at large, and effect healing. The common mode of origin, the similarity of the changes (though combined in different proportions) and the evident tendency of the inflammatory processes to protect and repair, justify fully the teaching that inflammation is essentially an adaptive, protective, and reparative process, a means of self-preservation. Yet it must not be forgotten that the mechanism of defence and preservation is far from perfect; the exudate may possess but little bactericidal power; the phagocytes may be powerless, or the bacteria may multiply freely within them; extensive destruction of tissue may occur before the virulence of the bacteria is neutralised; the "fixed" cells may form imperfect material for repair or multiply in excess . . . The inflammatory reaction does not respect the relative importance of the tissues. . . . . Hence, inflammation, though biologically an adaptive and preservative process, may appear harmful, requiring the intervention of medical art. Taking all things into consideration, we may conclude that inflammation is a reaction to local injuries, calling forth protective and reparative measures; but that it is an imperfect pathological adaptation, often leading to consequences that are dangerous *per se* and defeat its purpose." (Hektoen and Riesman. *Op. cit.*)

As this is true for the tissues in general, so does it equally apply to the dental pulp in particular.

It has been pointed out that passive or venous hyperæmia may soon pass into a condition of inflammation. A brief sketch of the *rôles* that are severally played by (*a*) the blood-vessels, (*b*) the colourless blood cells, (*c*) the exudate, (*d*) the "fixed" cells, and (*e*) the nervous system in this important condition must now be detailed.

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[1] Cohnheim: *Archiv für Pathol. Anat.*, vol. xv, 1867, xxlv., 1869.

[2] Metchinkoff: L'Immunité dans les Maladies Infectieuses, 1904. Pathologie Comparative de l'Inflammation, 1891.

(a) *The Blood-vessels.*—It is believed that the vessel walls are structurally altered during the course of inflammation to allow of and facilitate the emigration of the blood cells and plasma. Their endothelial cells are contractile (Klebs) and, according to Metchinkoff, mobile and phagocytic, and by frequently enlarging, cause an increased resistance to the vascular stream.

(b) The colourless cells play a fundamental part in the process, by passing into the perivascular tissues, as first pointed out by Dutrocht in 1828. Cohnheim laid great stress on this phenomenon. Detaching themselves from the marginal current, which they normally occupy on account of their low specific gravity, they become attached to the endothelial lining of the walls; and, as a result of a localised positive chemio-taxis produced by the diffusible products of bacteria, drugs, etc., emanating from the seat of the lesion, pass through the intercellular cementing substance. Leucocytic emigration is a complicated process, and varies with the nature of the cells actively engaged in it; thus there may be more eosinophiles than small mononuclear leucocytes, etc. The emigration is favoured by the dilatation of the blood-vessels and the contractility and mobility of the endothelial cells; and is determined by positive chemio-taxis by which the leucocytes advance toward the foci of greatest attraction.

The erythrocytes follow the white cells at greater or shorter intervals of time.

Phagocytosis is the action of certain leucocytes and wandering cells—with endothelial and "fixed" connective tissue cells to a limited extent—which occurs in the presence of pathogenic bacteria and other particles of matter. A kind of intra-cellular digestion takes place, alexins—protective bactericidal bodies—being formed, either by a process of secretion, or as Hardy believes, excretion, and the adventitious material being destroyed. The function is carried out by the neutrophile cells, and the polymorphonuclear leucocytes, and also perhaps the mononuclear leucocytes. Negative chemio-taxis—the antithesis of positive chemio-taxis—means the insensibility of phagocytes to and their actual repulsion from the toxins present in any particular part.

The inflammatory exudates possess also bactericidal properties, as shown by the experiments of Buchner, Nissen and others, and assist the phagocytes in their beneficial operations. All leucocytes are not phagocytes—eosinophiles are not; they possibly possess

excretory functions and may diminish the vitality of the micro-organisms.

(c) The inflammatory exudates contain more proteids than physiological lymph, also fibrin, fibrinoplastin, etc., and certain digestive ferments and peptones. The quantity is very insignificant in the pulp tissue; the serous and sero-fibrinous and fibrous exudates are small in amount and poor in quality; but the hæmorrhagic exudate, originated by the intensity of the primary lesion and due to an enormous emigration of the erythrocytes is very marked. After traumatic exposure of the pulp, during excavation of a deep carious cavity, for instance, where a thin layer of dentine remains in the floor, there is often a large flow of arterial and capillary blood, signifying extensive changes in, and injuries to the vessel walls and also a great quantity of hæmorrhagic exudate.

(d) *"Fixed" Tissue Elements.*—Retrogressive and progressive changes may go on side by side, but the former are more pronounced in the earlier stages of acute inflammation. Inflammation follows injuries that produce lesions not sufficiently great to induce complete necrosis and death of the part. No inflammation of the pulp is set up by cocaine pressure anæsthesia: it is anæsthetized merely. But in carious encroachments which give rise to an acute inflammation the pulp cells become greatly damaged and undergo extensive retrogressive changes. Necrosis and necrobiosis of the "fixed" cells and leucocytes occur. The odontoblasts at the site nearest to the lesion become fatty and degenerate; while further away they become "sheathed," and show signs of proliferation. This is probably an attempt on their part to heal the injury or prevent further damage from taking place, by warning the pulp, so to speak, of the oncoming dangers; and perhaps even to stimulate the dentine-depositing cells about them to functionate and produce adventitious dentine. These phenomena will be presently further alluded to.

(e) The sympathetic nervous system exercises a certain amount of influence in inflammation. Hyperæmia and exudation are interfered with by the uncontrolled action of the vaso-constrictors, the toxins are not removed, and repair of the injury cannot be proceeded with. If, on the other hand, the vaso-dilators exceed their functions, congestion takes place enormously or a strangulation of the vessels of the radicular regions chiefly soon leads to moist gangrene of the entire organ.

SPECIAL HISTOLOGY

Acute inflammation will be considered under two aspects:—
(*A*) When caries has not penetrated into the cavity, and (*B*) when it has penetrated.

A

Conditions Associated with Non-penetrating Caries

The odontoblasts, in the cervical regions, are enormously multiplied in point of numbers and layers. The cells themselves are not enlarged, but possess prominent oval nuclei which are much flattened laterally. Interposed here and there are small, hitherto
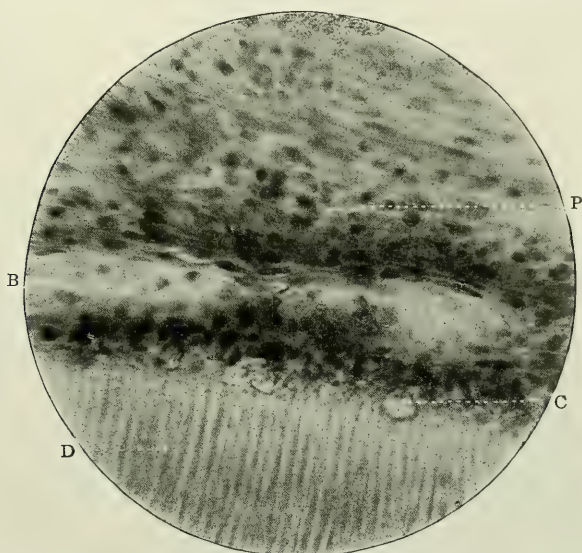
Fig. 166.—Acute inflammation of the pulp, with masses of calcoglobulin *in situ*. Prepared as in Fig. 163. D. Dentine; P. Pulp tissue; B. Enlarged capillary; C. Calcoglobular mass.

undescribed translucent globules, structureless and non-laminated, but similar in other respects to tiny calcospherite spherules (see Fig. 166). These are seen at the dentine border between the cells, and sometimes in Weil's layer. At the junction of the carious region with the primary or first-formed dentine the latest deposited dentine has, at its periphery, the globular appearances observed during developmental periods. It takes aniline dyes more deeply

FIG. 167.—An early stage of chronic (?)inflammation of the pulp. Prepared as in Fig. 163. Magnified 30 times. D. Dentine; P. Pulp; B. Enlarged blood-vessel; O. Odontoblasts gathered into sheaves.

than the normal dentine, from which it is highly differentiated. This new tissue may be called "adventitious" dentine—a term which includes several varieties previously noted. At the places where the carious tubules open into this freshly deposited layer, the odontoblasts are considerably shrunken, and pressed inwards towards the pulp. They are disposed in one, or at most, two layers, and their peripheral poles (dentinal fibrils) are greatly enlarged and swollen. The layer of Weil, of adult pulps, is most marked here. Micrococci and bacilli infect the newly formed tubes, and in some places expand them. And where tubular expansion has been effected, there the odontoblasts are absent, their places being occupied by a homogeneous mass of broken-down cells, with a few necrosed or necrobiotic nuclei scattered about. Active hyperæmia is distinguished by enlargement and tortuosity of the capillaries and arterioles, their engorgement with blood, and emigration of leucocytes (see Figs. 163 and 165).

As the thin sheet of adventitious dentine gradually widens out, the odontoblasts become elongated, remaining all the time in one layer, their fibrils, each with its individual enveloping tube, being of normal size, and stretching across the new dentine at fairly regular intervals. The cells themselves are sometimes gathered into sheaves—a condition frequently noticed in degenerative changes (see Fig. 167). With the widening of the sheet of dentine, they become smaller and shorter, and diminish rapidly in number, until they disappear altogether.

Meanwhile the adventitious dentine presents the well-known appearance of areolation almost identical with that of the interglobular spaces. This areolar adventitious dentine has been described on p. 72. The structure of the pulp itself at this place is of the homogeneous character already noted. Thus the vessel walls are changed, the endothelial cells being enlarged and projecting into the lumina of the capillaries, and undergoing mitosis. The active movements of the emigrated leucocytes, which have already passed through the intercellular cement substance of the vessel walls, are directed to certain areas in the tissue through the influence of positive chemio-taxis set up by the diffusible products of the bacteria (bacteri-proteids) in the tissues, and of other substances. The serous or sero-fibrinous exudation is necessarily slight; and the fixed connective tissue cells of the pulp, having undergone cytogeny, suffer from retrogressive changes, and, becoming necrotic or necrobiotic, show signs of cloudy swelling, disintegration and death.

The dentine which fills the cornua of the pulp exhibits irregular formations, as if deposition had taken place in a hurried manner. Not only are nucleated cells with long processes imbedded in the hard mass, but large lacunal spaces are frequent, each containing micrococci which have entered *viâ* the tubules of the primary dentine. In some instances this cellular dentine somewhat resembles the structure of sponge.

A bacteriological survey of the same specimens of hyperæmia and early stages of the lesions which Rothmann[1] has designated
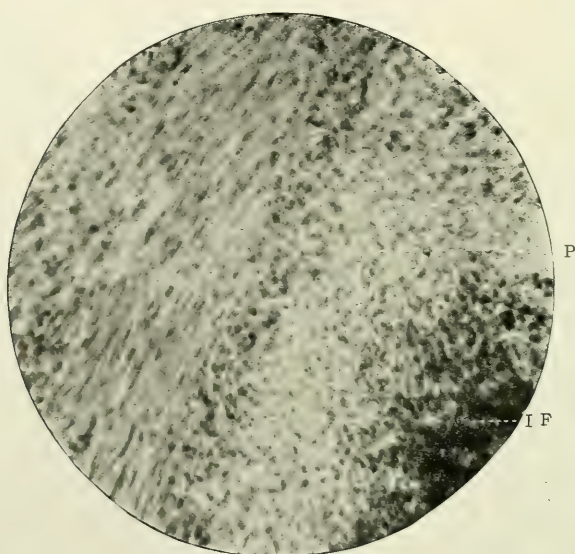
Fig. 168.—Acute inflammation of the pulp. Stained with Ehrlich's acid hæmatoxylene and eosin. Shows the inflammatory products attracted to a focus of inflammation. P. Leucocytes and proliferated connective tissue cells; I.F. Inflammatory focus.

"Partial acute pulpitis," and Wedl[2] "*Pulpitis acuta partialis*," furnishes one with some valuable particulars as to the probable distribution of the micro-organisms in the pulp and surrounding tissue. Miller[3] has isolated, cultivated, and named the most important of the cocci and bacilli; here is an opportunity of describing the probable routes of their invasion of the pulp itself.

The micro-organisms, after their introduction into the pulp

[1] "Patho-Histologie der Zahnpulpa und Wurzelhart," 1889.
[2] "Atlas zur Pathologie der Zahne," pp. 68, 69, 1893.
[3] "Micro-organisms of the Human Mouth," 1889.

cavity, are believed to make their way in chains, groups, or masses to the spaces between the odontoblast layer, the dento-genetic and ordinary pulp cells on the one side, and the border of dentine on the other; and also to the interpolar (interfibrillar) spaces, and the intercellular intervals. Thence they travel apparently to the basal layer of Weil, although here they are not congregated in such large or such numerous masses. Whatever their point of entrance, they soon pass to some considerable distance along the line of junction of the hard and soft tissues.

Further, they are found in the substance of the pulp proper, chiefly arranged along the walls of the blood-vessels, in their interiors (when empty), and in the perivascular tissues. Infection of the nerve fasciculi most probably does not take place. The micrococci predominate largely over the rod-shaped organisms. The central and peripheral portions of the adventitious dentine are crammed with micro-organisms, but when the odontoblast fibrils with their sheaths cross the areolations of this new deposit, no bacteria can be found.

From these investigations, therefore, it will be seen that, as a result, one is unable to coincide with Arkövy's theory of the phagocytic function of the odontoblasts.[1] They certainly possess a granular appearance, but a search for any micro-organisms which have become incorporated in the substance of their cytoplasm or nuclei is attended with negative results.

If the course of the disease is progressive, inflammatory foci appear. These consist of proliferated connected tissue cells (macrophages), pulp cells, and lymphocytes or polymorphonuclear leucocytes which have escaped from the numerous enlarged capillaries, all having been attracted together by positive chemio-taxis. The foci are very pronounced, commence at first in one or both of the cornua of the pulp opposite the carious dentine, and, as a rule, ultimately suppurate and form localised abscesses (Figs. 169 and 170). Rapid destruction of the pulp ensues, and the undermined dentine finally gives way in the majority of cases.

Sometimes a certain amount of fibrification of the cells lying in the immediate vicinity of the abscess occurs, and what might be termed a rudimentary abscess wall is developed. One is led to believe that this specialisation and grouping of spindle cells is not merely fortuitous, but a deliberate attempt on the part of the pulp to heal the lesion. The condition is observed in cases of chronic caries, the adventitious dentine being then deposited in layers, and presenting

[1] See *Journal of Brit. Dent. Assoc.*, vol. xv., p. 602.
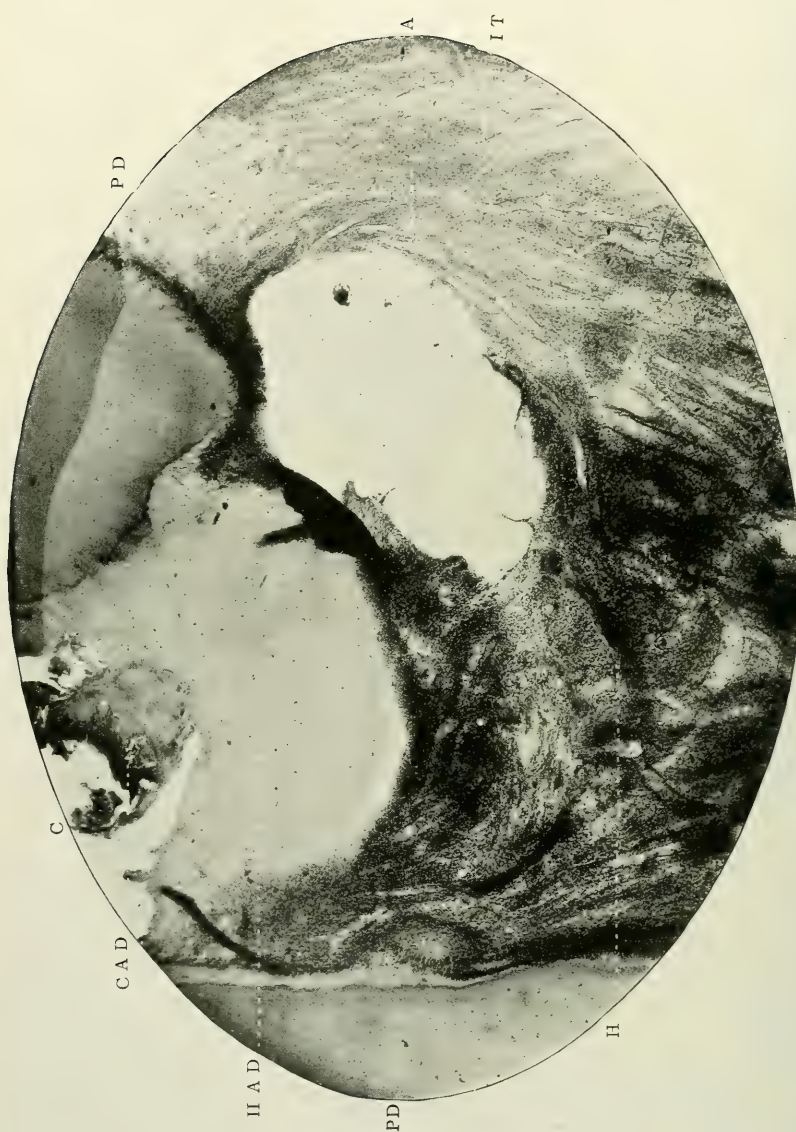
Fig. 169.—Vertical section of human molar tooth with inflammation of the pulp. Prepared by the Author's process. Stained with Ehrlich's acid hæmatoxyline. Magnified 50 times. P.D. Primary dentine; C. Carious cavity; H.A.D. Hyaline adventitious dentine; C.A.D. Cellular adventitious dentine; I.T. Inflamed pulp tissue; A. Abscess cavity, the pus corpuscles having been washed away; H. Intense hyperæmia and inflammation of the pulp.
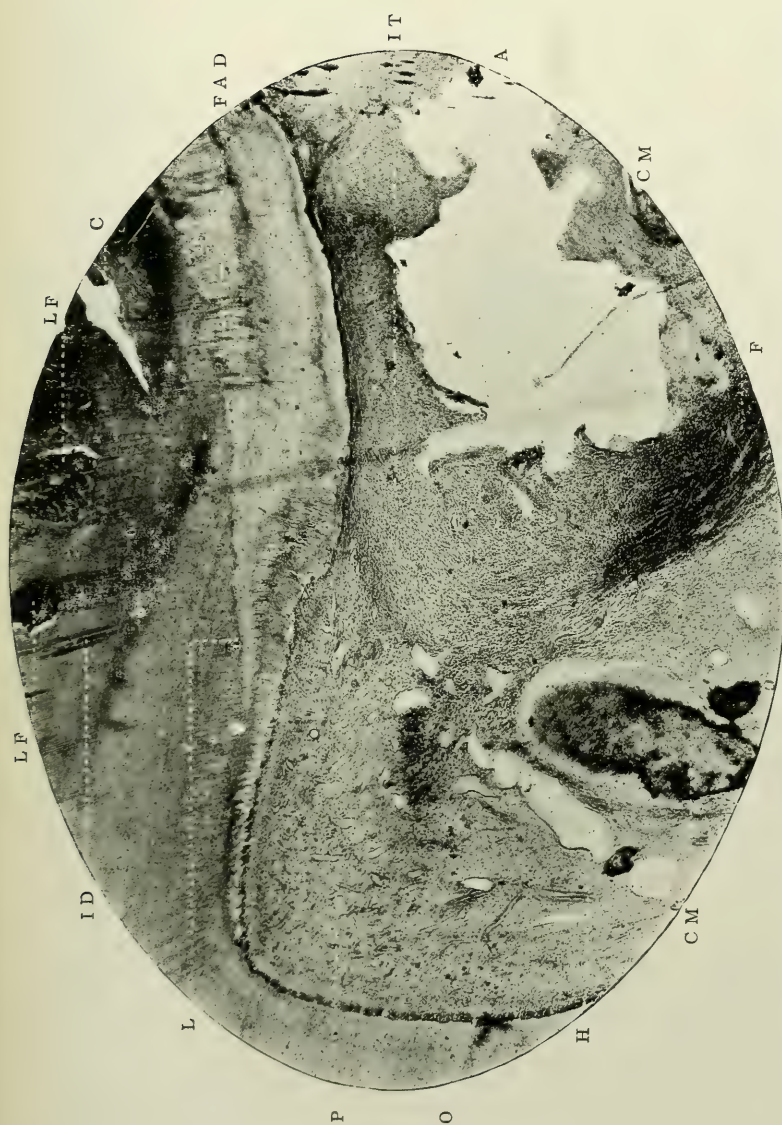
FIG. 170.—Vertical section of human molar tooth, affected with caries and inflammation of the pulp. Prepared, stained and magnified as in Fig. 169. c. Carious cavity in the primary dentine; I.D. Primary dentinal tubules infected with micro-organisms; L.F. "Liquefaction foci;" L. Original limit of the pulp cavity; F.A.D. Fibrillar adventitious dentine; P. Pulp tissue, apparently but slightly affected by the inflammation; O. Original odontoblasts; I.T. Acute inflammation of the pulp tissue; A. Abscess cavity, the pus corpuscles having escaped; F. Early attempts at the formation of the walls of an abscess sac; H. Hyperæmic blood-vessel; C.M. Mass of calcoglobulin.

a characteristic fibrillar structure. On the border-line of the hard and soft parts, the connective tissue structure of the dentine matrix is well brought out. Islands of semi-calcified material in the body of the pulp suggest that they are nothing more nor less than calcified bundles of connective tissue fibres mixed with cells; the process of their formation being analogous to that of intra-membranous ossification of bone (see Fig. 170).

B

Conditions Associated with Penetrating Caries

The exigencies of the scope of this book afford these notes opportunity of speaking of no more than two phases of one of the com-
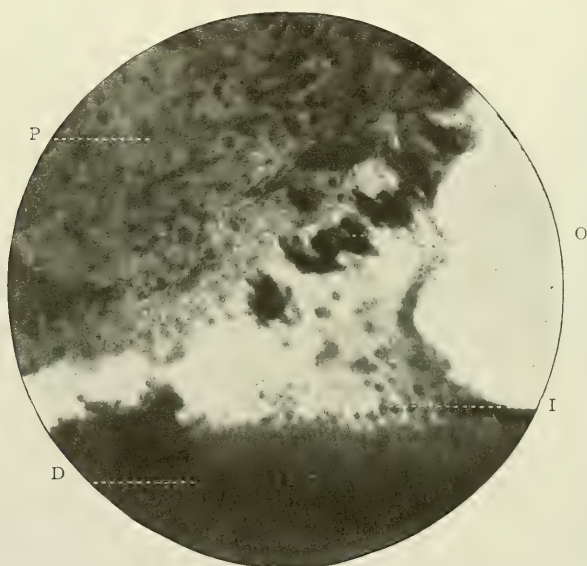
Fig. 171.—Inflammation of the pulp. The odontoblasts have been torn away from the surface of the dentine, to show the presence there of the proliferated connective tissue cells, leucocytes, and other products of the inflammation. Prepared by the Author's process. Stained with Ehrlich's acid hæmatoxylene. Magnified 180 times. D Dentine; P. Pulp tissue; O. Odontoblasts; I. Inflammatory cells, etc.

monest conditions found in the mouth, viz., idiopathic exposure of the pulp.

In sagittal sections of teeth affected by acute caries, which has terminated in acute inflammation and partial suppuration of the pulp, it is obvious that the cells appear degenerate altogether.

Connective tissue cells are broken down, the pulp cells have become changed into indifferent cells with large square nuclei, and escaped leucocytes crowd the tissues. Even the odontoblasts themselves at the cervical region are metamorphosed into short cells with rounded nuclei, and at the coronal part are opaque, and seem to have undergone fatty or mucoid degeneration (Fig. 172).

Finally, at the periphery of the pulp the small globular deposits, already mentioned, are found. The nerve bundles have lost their definite structure, and though retained in position are evidently less
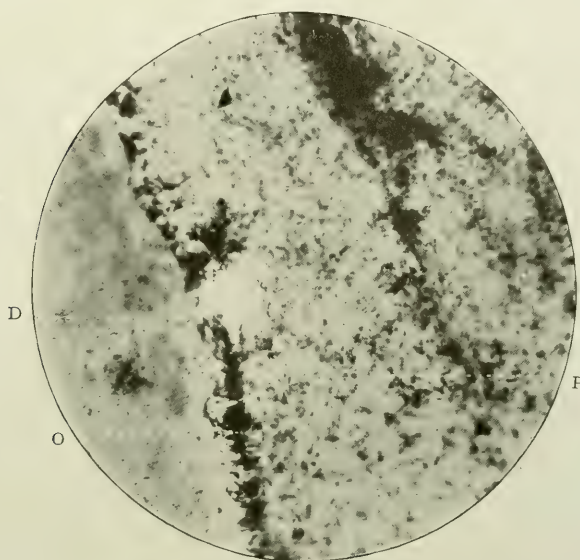
FIG. 172.—Acute inflammation of the pulp. Prepared and stained as in the preceding figure. Magnified 80 times. D. Dentine; P. Pulp tissue crowded with inflammatory products; O. Odontoblasts.

translucent and more disorganised. There are no clear evidences of fatty degeneration in the sections under notice, although Wedl[1] describes this as existing in his sections of acute purulent pulpitis. The tissues are greatly condensed at the margins of the abscess cavity, the cells being short and fusiform, and interlacing closely with each other. The blood-vessels are hyperæmic, and micrococci and bacilli are abundantly distributed throughout the tissue.

The last condition which will be here considered is that of a phase of acute partial suppurative inflammation of the pulp, in which

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[1] *Op. cit.*, p. 71.

that organ has been subjected to the devitalising action of arsenious acid for a period of not less than twelve hours.

In addition to appearances which denote the intensity of the inflammation—hyperæmia, marked cellular infiltration, suppuration and other changes common to acute inflammations in soft tissues— a prominent feature is a large special form of dentinal deposit which is situated at the base of the carious opening into the pulp chamber. This is cellular and hyaline adventitious dentine previously described on pp. 72 and 74.

This particular form of cellular or hyaline adventitious dentine does not occur solely in acute inflammation of the pulp, it is also seen in chronic inflammation with hyperplasia ("polypus") near the lower portion of the pulp cavity. In this case it may be accompanied by new dentine which has a pronounced laminar structure.

In conclusion, a study of the patho-histology of these lesions leads one to the following deductions:—

(1) That nearly every degree of dentinal change is attended with hyperæmia, and cell proliferation in the pulp tissues, and generally speaking, the formation of adventitious dentine:

(2) That the latter may have its origin as a conversion or secretion of the dento-genetic cells, producing on the one hand the areolar or laminar or hyaline varieties, when the formative cells alone happen to be concerned; on the other, the fibrillar or cellular forms when odontoblasts or connective tissue cells are by chance incorporated in the deposit:

(3) That the new dentines, by a system of extension from the affected areas, may be just as much subjected to the peptonising action of micro-organisms as the primary dentine of the tooth.

Chronic Inflammation

This is sometimes one of the sequelæ of acute inflammation. The pathological processes are essentially the same. The changes, however, are continuous, thus: The dilated vessels remain in a condition of dilatation and lose their tone, leucocytes continue their work of emigration through the vessel walls, and the original tissue cells still further proliferate.

Etiology.—The causes are similar to those of the acute conditions; but certain general systemic diseases such as rheumatism, gout, hydrargyrism, etc., are believed to be powerful, predisposing, and, in some cases, exciting causes.

HISTOLOGY

It is nearly always associated with the formation of large masses of calco-globulin nodules in the pulp, and may go on until the whole of the pulp tissue becomes converted into a hard calcareous mass.

When there is exposure of the pulp, these deposits of calco-globulin are always seen in the region of the inflamed area. They are irregular in shape, and often attain a large size, and fill up the pulp tissue. Sometimes these dentine masses extend throughout the pulp like long rods running parallel with the walls of the pulp chamber. The pulp, when removed from its cavity, is found to be stiffened and hard from the presence of these calcified rods. They are seen in the immediate neighbourhood of the blood-vessels and nerve bundles.

When stained, for microscopical purposes, they behave like the dentogenetic zone in developing teeth—that is they take fuchsine, hæmatoxylene, carmine, and other stains very intensely.

If there is an extensive exposure of the pulp, the tissue often undergoes productive inflammation, and becomes hyperplasic, and the condition known as *polypus* is found.

Hyperplasia of the pulp is a chronic productive inflammation, in which the redundant material formed is extruded through the opening at the base of the carious cavity. At first small, it slowly increases in size, until ultimately a large soft fungating mass fills up the cavity, and sometimes overhangs its edge. It must be diagnosed from hypertrophy of the gum, the two points of difference being usually easily determined.

Hyperplasia of the pulp is probably caused by the irritation of sharp dentinal margins of the pulp cavity, which become slowly absorbed as the condition advances.

The *patho-histology* is interesting. The mass consists briefly of (i) a superficial epithelial layer, (ii) a stroma or frame-work of strong fibrous tissue, (iii) supporting the large granulation-like cells and blood-vessels, which constitute the greatest parts of the growth.

(i) The superficial epithelial covering consists of a stratified layer or layers of large squamous epithelial cells. The occurrence of squamous epithelium in the surface of a tissue which does not normally contain any epithelial cells is remarkable. This auto-plasty was formerly believed to be due to a form of skin-grafting brought about by the transference to the free surface of the exposed pulp of portions of epithelial tissue from the mucous membrane of

the cheek, during the act of mastication. The truth is that the
surface cells of a portion of gum which overlaps an edge of carious

FIG. 173.—A large fungating hyperplasia of the pulp overhanging the edge of
a carious cavity. Stained with hæmatoxylene and eosine. Magnified 230
times. D. Normal dentine; E. Edge of carious cavity, C. Cementum; P. Pulp
tissue; E.A. Epithelial-like areas in pulp.

dentine rapidly proliferate as a result of the mechanical irritation,
and spread over the exposed portion of the pulp to which they
finally become firmly adherent.

The periphery of the growth may present one or two different structures. (1) It generally consists of very flattened non-nucleated cornified squamous epithelial cells, with little intercellular cement substance. (2) The periphery may closely copy the ordinary epithelial characteristics of the mucous membrane of the mouth and gums, that is to say, its sub-mucous surface, is thrown into innumerable folds or papillæ.

In the first instance several thin layers of epithelial cells, arranged as strata, sometimes may be found. The outer layers are composed
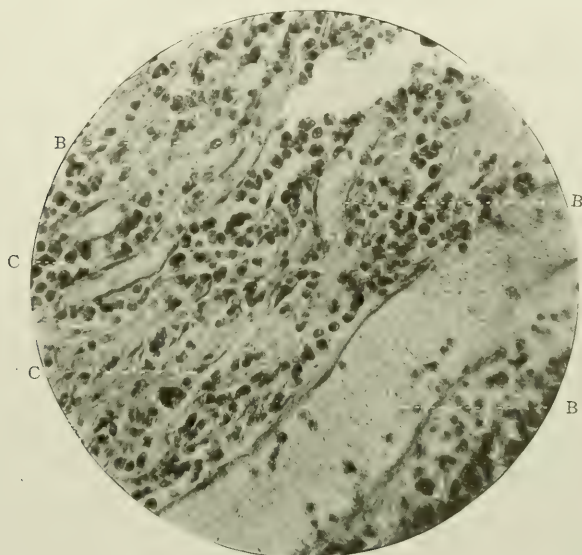
FIG. 174.—Chronic inflammation of the pulp, with hyperplasia of that tissue (so-called "polypus"). Prepared as in the preceding figure. Magnified 250 times. B. Blood-vessel crowded with corpuscles; C. Nuclei of the inflammatory cells.

of flat cells, the inner of ovoid cells, generally two or three rows. Each cell is granular. Finally, there appears the *stratum Malpighii*, where the cells are smaller and columnar in shape with oval nuclei. These rest on the papillæ, a sort of basement membrane dividing them from the general sub-mucous tissues beneath. The *papillæ* found in the second instance are conical elevations projecting into the layer of the *rete Malpighii*. If they are undivided they are known as *Simple;* if they are beset with smaller papillæ they are called *Compound*. They consist of compact fibrous tissue.

The rest of the "polypus" proper consists of a fibrous stroma,

in which the fibrous tissue is very pronounced, firm, and interlacing in all directions. In the meshes of this reticulum are found large granulation cells, some round, some square, and each having a large nucleus (see Fig. 174).

The capillaries terminate at the periphery, and run amongst the meshes of the stroma. At its attachment to the pulp cavity, the fibrous tissue is arranged in strong bands which stain a deep yellow colour with picric acid. The blood-vessels in this portion are scanty, but larger than those seen at the periphery.

Here also rounded masses of laminar adventitious dentine are found. They have a laminated nature, one or two small cells occasionally occupying the interior of the pulp nodule.

In the pages of *L'Odontologie* of September 15th, 1902, Pont describes an extremely curious neoplasm of the pulp which is almost unique in the annals of Dental Pathology.

Under the title of "Note sur un cas de Tumeur de la Pulpe dentaire sans carie de la dent," an account is given of this anomaly, occurring in the second maxillary right premolar of a man of 35 years. He complained of pain. An enamel chisel removed the occlusal surface of the tooth which was perfectly sound, and revealed a "soft, deep red, easily torn, but not painful growth of the pulp, which had produced absorption of the cavity walls." It was extirpated easily, the operation being unattended by much hæmorrhage.

A histological examination having been made by Dr. Charvet, of the Faculty of Medicine, of Lyons, this gentleman reports that it was not a tumour properly so-called, but probably a simple inflammatory condition of the pulp. He wrote as follows:—

"La pièce à examiner, n'a nullement le caractère d'une tumeur. Sur les coupes colorées au picro-carmin, on trouve des traces de travées sclèreuses, et dans tous les points, des nappes de cellules inflammatoires; ailleurs enfin des hémorragies interstitièlles plus ou moins étendues. Nulle part il n'y a de masses néoplasiques, ou pouvant y faire songer. Il semble s'agir d'un bourgeon charnu inflammatoire en voie d'organisation sclèreuse."

Infective Gangrene of the Pulp

These chemical and biological changes proceed very rapidly. The first stage begins as a *post-mortem* change, and does not necessarily depend upon the presence of *Schizomycetes*, the fact being that both the liquid and solid constituents of the pulp immediately after

its death are capable of destroying many micro-organisms. The changes partake more of the nature of a chemical dissolution than a result of bacterial infection, and are dependent upon the action of the unformed soluble ferments found in the pulp, as in other soft tissues. These soluble ferments or enzymes are present in all living tissues, and have much to do with the processes of metabolism. Thus albumen becomes converted by these enzymes into peptones and hemi-albumens.

Pathogenic bacteria are capable of developing in the soft tissues and bringing about the changes just mentioned. But in order that they may develop sufficiently to produce these chemical changes in sufficient amount before they themselves are destroyed, there must be some local focus of disease or area of chemical decomposition present, which becomes largely invaded by pus-forming micro-organisms.

It is therefore obvious, that if the balance between the biological actions of the invading and invaded forces is maintained in equilibrium, simple death of the pulp will occur; that is, its general functions will cease, its physiological resistance to disease or injury will be in abeyance, its powers of undergoing progressive or further retrogressive metamorphoses will be terminated, and it will remain, perhaps for many years, an inert, innocuous, ineffectual remnant of its former self—pale and shrunken. The hard parts surrounding it will, at the same time, be affected by the loss of nutrition, and the enamel—probably through changes in the subjacent dentine, certainly not through actual alteration of its own structure or chemical composition—will eventually become dark and lustreless. This is what happens in the case of a tooth which has been "killed" by a blow on the mouth.

On the other hand, if the bacterial infection is great and the albuminoid bodies thus produced are voluminous in amount, toxic enzymes result. These are very active poisons, which give rise in a short space of time to the usual chemical products of decomposition, viz., carbonic acid, ammonia, sulphuretted hydrogen, and certain other salts and water. The evolution of these is dependent upon the access of oxygen, heat, and moisture.

According to Buckley (*Trans. Fourth International Dental Congress*) the chemical changes of putrefaction are those of hydration, reduction, and oxidation. In the first, there is a taking-up of one or two molecules of water, in the second, a breaking-up and decomposition by nascent hydrogen, and in the third, the

formation of carbonic acid, acetic, nitrous, nitric and similar acids. Other substances manufactured simultaneously, in varying degrees, are globulins, toxic enzymes, peptones, the nitrogenous amino-acids, leucin and tyrosin, the nitrogenous amines, methylamine, propylamine, etc., and organic and fatty acids such as formic, propionic, butyric, valerianic, palmitic and other fatty substances and also
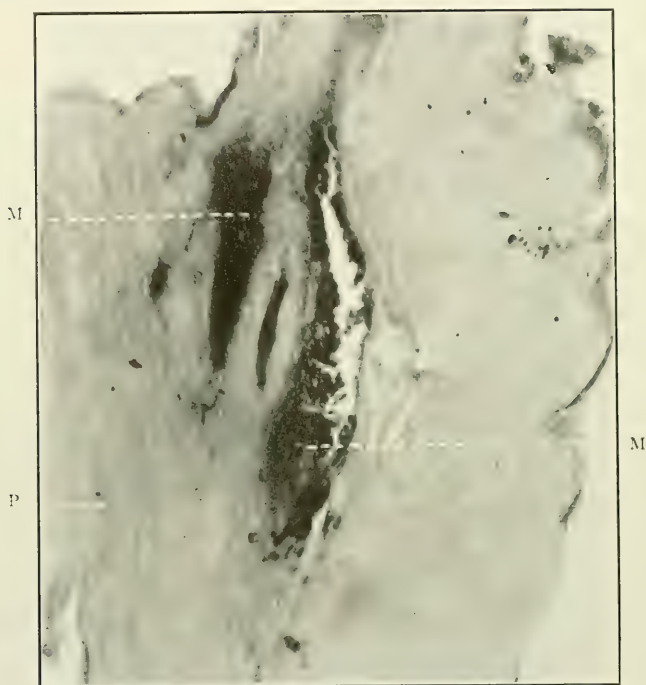
FIG. 175.—Vertical section of infective gangrene of the pulp removed from its containing cavity. Stained by Gram's method. Magnified 45 times. M. Dense masses of micro-organisms; P. Non-infected pulp tissue.

putrescine ($C_4H_{12}N_2$), and the isomers cadaverine and neuridine ($C_5H_{14}N_2$). These latter become quickly converted into ammonia and its derivatives. Thus is the pulp destroyed, and it is probable that the contents of the dentinal tubes are also similarly filled with the end-products of decomposition.

The Pathogeny of Gangrene of the Dental Pulp

A frequent termination of inflammation, whether traumatic in origin or not, is gangrene, or mortification of the pulp. The

patho-histology of this condition has been studied by Dr. Fritz
Schenk, of the Dental Department of the University of Vienna.
An interesting article from his pen appears in the *Oesterreichisch-
ungarische Vierteljahrsschrift für Zahnheilkunde*, for March, 1902.
The following are some of his important observations.

"At the apex of the pulp a gangrenous mass, entirely devoid of nor-
mal tissue elements, is found in the earliest stages of the disease.
A line of demarcation cuts this off from the healthy portions of the
organ, and contains disintegrated cells. In the gangrenous re-
gion, as has just been stated, no cells or tissue fibres can be recog-
nised, but in fresh gangrenous patches fat globules, formed by a
process of dissolution, appear. Micro-organisms are present in
this situation, according to Miller, Arkövy, and Scheff. This con-
dition corresponding to Rothmann's "*Pulpitis gangrænosa chronica*,"
has also been investigated by Witzel and Arkövy. This gan-
grenous patch is surrounded by the "line of demarcation." The
rounded contours of nuclei may sometimes, especially if a solution
of eosine be used as a staining reagent, be noticed at the borders of
this patch. Their chemical characters may be altered, and indi-
cate a "transition stage" towards final dissolution. In the neigh-
bourhood of the non-gangrenous portion the nuclei decrease in
number; and this together with the passive hyperæmia present con-
stitutes the chief signs of the "line of demarcation."

"In the remaining tissue, which generally borders on the
gangrenous portion, and is in course of inflammation, there is one
factor to be especially noted, that besides migrated red blood-
corpuscles in the tissue, the vessels appear enlarged, sometimes
filled with coagulum. The appearance may be seen, both in longi-
tudinal and in transverse sections, only at the line of demarcation;
nearer to the gangrenous portion the vessels are entirely missing.

"The termination of an inflammatory process in the pulp often
leads to gangrene, which does not cover the whole or major portion
of the pulp, but generally only includes at first the distal parts.
From this point it proceeds towards the centre. For the conclusion
of the process there is needed a longer or shorter period; therefore
this process has been correctly designated *Pulpitis gangrænosa
chronica*.

"Contemporaneously with this course, certain phenomena also
occur in the tissues, which lead to the destruction of the pulp.
These processes likewise develop comparatively slowly.

"In the first place one process regularly occurs in such pulps,

the importance of which in all inflammatory conditions is to be specially emphasised. The red blood-corpuscles secede from the coagulum contained in the enlarged vessels. One observes them lying everywhere freely about in the tissue. Especially remarkable is this at the line of demarcation, where the colouring can be seen macroscopically or with the magnifying glass.

"When the nutrition of the protoplasm of the cellular tissue reaches abnormal conditions, as is the case with inflammation of the pulp, it may easily happen that in the protoplasm there supervenes coagulation of its albuminoid constituents, which may be of a fibrino-plastic nature. This view in regard to coagulation (Alex. Schmidt) is here adopted.

"If coagulation of the protoplasm has once occurred, every function within it which is necessary for the preservation of the cellular tissue is made difficult in performance, and the cell must therefore go through a retrogressive metamorphosis, which finally leads to degeneration. Furthermore, the general law is also of effect, that the protoplasm of the tissue elements has a certain connection with their nuclear formations.

"Consequently, both morphological constituents of the cell— the protoplasm as well as the nucleus—are indivisible and independent, in order to manifest the vital function of the cell. The nucleus is a necessary attribute of the cell, and becomes its dominant constituent, inasmuch as it governs its growth. It undergoes mitosis, and influences the histological differentiation. The nuclei produced by the fission resemble the mother-nucleus (O. Hertwig). Portions of protoplasm, without nuclei, may maintain themselves for a time, however, without multiplying.

"Many portions of protoplasm are incapable of fulfilling their physiological functions as soon as the nucleus has disappeared from them. Leucocytes, whose protoplasm possesses qualities of life and movement, but whose nuclei are not divisible, die, and remain in the living organism as pus corpuscles.

"The protoplasm which is fitted to absorb material from the surroundings, and to make corresponding use of it, loses all the qualities peculiar to it through the coagulation of its albuminoid bodies. Through this coagulation it not only loses the power to absorb materials and to convert them, but, furthermore, it is no longer capable of phagocytic functions.

"In this manner it can be easily explained that in chronic gangrene of the pulp the first impulse for its origin is given by the coagulation

of the protoplasm of the cellular tissue. It is the primary affection, in the wake of which follow the other appearances of gangrene.

"The alterations of the nuclei appear only when the protoplasm has lost its vitality through the coagulation of the albuminoid bodies. By this means the relationship between the nucleus and the protoplasm entirely ceases. The nuclei become smaller, and gradually lose more and more of their karyoplasm, and decrease in size until they have shrunk to dot-shaped remnants of the chromatic substance. Finally these also disappear, and one only sees circumscribed, light, uncoloured portions in the decayed granular gangrenous substance, which possibly still exist as the last remains of the former nuclei. It is true that the cell nuclei exhibit a longer power of resistance during this pathogenic process than all the other tissue constituents of the pulp, but at last their individual recognisable morphological elements disappear, and with them the dignity of being able to act in the regeneration of the elements."

CHAPTER VI

INJURIES OF THE DENTAL PULP

MICROSCOPICAL ELEMENTS FOUND IN: (i) Methods of healing after wounds of the pulp.

GENERAL CHARACTERISTICS

Of all the morbid conditions usually associated with the dental pulp it is generally conceded that those reparative processes which occur after injury or during the course of a disease of that organ rank first in interest and importance. A study of the methods by which nature attempts to repair or heal a lesion is, on careful consideration, one of the most fascinating subjects that can engage the attention of the pathologist. If this is so generally, in the great domain of general surgery and pathology, how much more interesting to dental surgeons, must be a study of a like nature when connected with that most delicate organ the dental pulp?

It is not at all surprising that this is a structure which possesses great recuperative powers, and is constantly exercising its functions in this respect by undergoing repair. Very seldom indeed can a lesion of the soft or the hard parts of a tooth occur without a corresponding attempt—more or less successful—on the part of the pulp to ward off its attacks. For in the exercise of its highest functions it is concerned with the maintenance of the vitality of the tissues in the centre of which it is placed. Hence any invasion by disease, or the occurrence of an accident is succeeded by a resistance which in many cases is highly satisfactory, and the tissues are not devitalised. Probably nearly every dentinal change is accompanied by some healing process of the pulp.

The restoration to a normal condition of pulp lesions partakes of the characteristics both of those of the soft parts, like a wound of the skin or other vascular tissue, and those of bones, as in the case of fracture. Instead of a permanent or definitive callus being formed in the pulp cavity, the conditions are modified through the anatomical peculiarities of the part: and, as a result, the various kinds of adventitious dentines, already fully described, occur. It is unnec-

essary to lay stress on the fact that here, as elsewhere, the process is essentially similar. Osteoblasts, which are the great factors in the production of bone, are not found in the pulp; but the dento-genetic cells, with which the tissue is freely supplied, do similar work. When the pulp is nearly exposed, for instance, they combine to repair the damage done by caries. Examples of "dentine of repair" are often seen. In traumatism, too, the process is exactly the same as when a tissue has been wounded, and has been kept in an aseptic condition, and properly protected from certain infective processes. In these cases the method of repair is known as "healing by the second intention," or granulation. A third, but exceedingly rare class of cases, where a tooth, having been fractured, and the parts kept at rest until union has been effected, supplies an example of "healing by the third intention." Cemental or dentinal union is only possible, it is obvious, when pulp or periodontal membrane, or both, have received an injury which has not destroyed their reparative powers, but which has induced a slight or sub-acute form of inflammation, and thus stimulated the active cells to perform their functions.

These notes, however, are not intended to generalise altogether, but to particularise, and to sum up what is known of some of the healing processes in the pulp.

It will be convenient, then, to consider the subject from several points of view;—(1) When a minute area of the pulp tissue has been injured; (2) when a large surface has been traumatically exposed; (3) ordinary exposure of the pulp by the action of caries; (4) fracture of teeth with or without impaction of the fragments; and finally (5) in cases of non-exposure of the pulp.

HISTOLOGY

I

When a minute area of the pulp has been injured.

It is not difficult to conceive of a simple traumatic inflammation of the pulp being set up when a small traumatic exposure has been made, which has been immediately followed by the penetration (and therefore wounding) of the soft tissues by means of a fine point like that of a bristle. The picture that can be drawn of this rather hypothetical state of things is quite clear, from one's knowledge of wounds generally.

The capillaries, together with the small arteries and veins in the coronal region, would be divided, causing momentary hæmorrhage,

which soon ceases in consequence of stasis and coagulation of the blood. Dilatation of the vessels in the neighbourhood with relaxed flow of their blood-currents next supervenes, leucocytes and *liquor sanguinis* escape from their walls, and the cellular elements around undergo sub-division and proliferation. Lymph in minute quantities is poured out, and contains fibrin and blood corpuscles; the serum becomes absorbed and approximation of the divided surfaces results. Outside the region of the wound the vessels are dilated, but the rate of the flow of blood is increased. Shortly after the
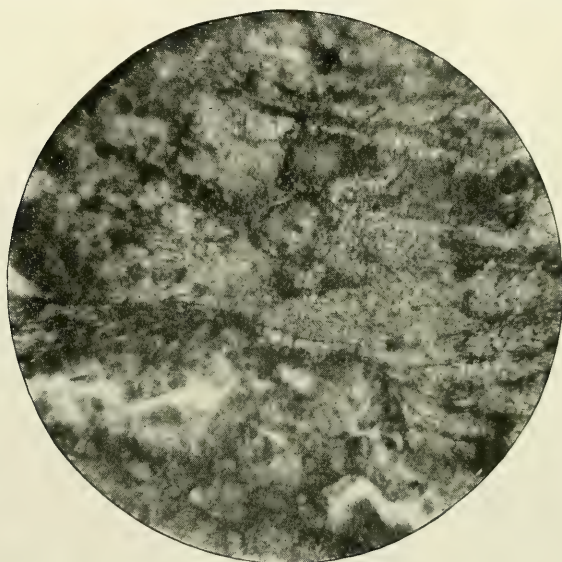
FIG. 176.—Coagulation necrosis and fatty necrobiosis of the superficial portions of the pulp occurring after a chemical injury of twelve hours' duration, following a sub-acute inflammation of the pulp. Stained with hæmatoxylene and eosine. Magnified 200 times.

injury, the exudate, which has coagulated, more or less, is removed and replaced by small round cells derived from the infiltrating leucocytes and proliferating connective-tissue cells, and thus adhesion of the separated parts takes place. Finally, delicate loops of new capillaries emanating in their origin from the old vessels spread across the parts, and anastomosing, restore the vascularity of the tissues, and repair the lesion by the production of fibrous bands and ultimately a small cicatrix.

Such a condition of things could only be accomplished when the pulp was only slightly injured; when the foreign body producing the

injury was not septic; and when the cavity in the dentine had been treated by strictest antiseptic measures.

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When a large area of the pulp surface has been traumatically exposed, as occurs during fracture of the crown of a tooth in an attempted extraction, regional hyperæmia occurs and inflammatory symptoms are set up. Many interesting microscopical structures are now brought to light.

If sections are made within *twelve hours* of the accident—or rather if the preparation of the tissues for section cutting is begun within twelve hours—the pulp will be found to be crowded with small round cells. These comprise the infiltrating leucocytes and proliferating connective-tissue cells. The odontoblasts are profoundly altered in shape, being flattened and compressed on to the dentinal walls through the swelling and exudation of the inflammatory processes. They have also undergone mytosis and are numerically multiplied. The vessels are actively hyperæmic, and in the radicular portions of the pulp are largely thrombosed. On the free surface of the pulp an attempt at healing has occurred, organisation of the exudative fluids and materials having taken place. Blood cells, escaped from the broken coronal capillaries, are caught and retained in the meshes of a fine fibrous stroma of new formation. The cells are very greatly disintegrated, and probably many of them have been converted into pus corpuscles. Probably, too, coagulation necrosis and fatty necrobiosis of the superficial tissues have, to a certain extent, taken place, as there are some appearances of fibrin formation as well as degenerative lipogenesis in the midst of the tissues (see Fig. 176). It is unlikely that micro-organisms would produce liquefaction of the parts in so short a space of time as twelve hours, especially as a thick blood-clot would have protected the free surface from the oral secretions. It is almost certain that if liquefaction of the necrotic tissue and the solid exudates has occurred it is due to proteolytic enzymes.

On examining sections where a similar lesion had occurred *seven days* previously, but few fresh changes are noticed. The free surface is covered, as before, with many layers of disintegrated cells, including pus cells. Traces of the original inflammation have more or less disappeared and new capillaries abound, freely vascularising the part. But one notices everywhere, and especially in

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the cervical and radicular regions, new deposits of soft calcific material in the midst of the tissues. These have the same appearance as that shown in Fig. 170. The laying down of calco-globulin is a favourite method of repair on the part of the pulp. Whence
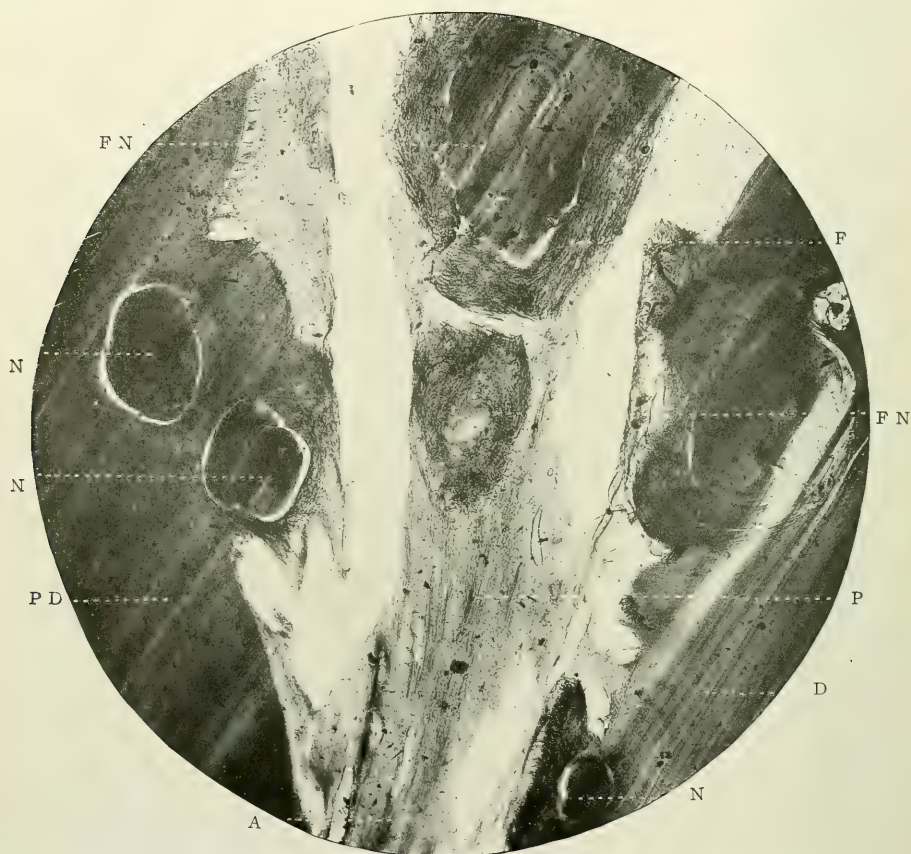
Fig. 177.—Freshly deposited calcific nodules in the pulp, showing the method of formation. Prepared by the Author's process. Stained with borax-carmine. Magnified 50 times. P.D. Primary dentine of the tooth; N. Formed and calcified dentine nodules incorporated in the walls of the pulp cavity; F.N. Nodule formed but not yet calcified; F. Connective fibrous stroma with dentogenetic cells, about to form the calcified masses; P. Pulp tissue with nerve bundles and blood-vessels; A. Pulp near apical region.

come these new masses? How are they brought into existence? What laws govern their formation? These are questions which one finds considerable difficulty in answering satisfactorily. It is unlikely that they are due to the local enzymes of micro-organisms.

or that they are found congregated around masses of bacteria. They are not always dependent on being closely approximated to vessel walls, for they are seen in other situations. Black[1] inclines to the belief that the formation of these masses is due to a condition of congestion or venous hyperæmia of the pulp. He compares them to the phleboliths found in varicose veins, and suggests that in these congested veins there may exist the three conditions, or factors, necessary for the production of calcospherite globules,
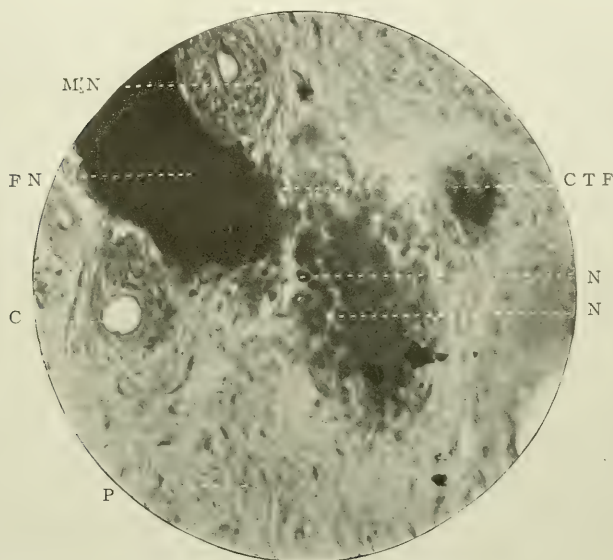
FIG. 178.—An early stage in the formation of nodules of calcific material in the pulp. Shows methods of formation. Prepared as in the preceding figure. Stained with orange rubine. Magnified 250 times. F.N. Nodule undergoing calcification; N. Nuclei of the formative cells; C.T.F. Connective tissue fibres; M.N. Myelinic nerve bundle; C. Capillary; P. Pulp tissue proper.

viz., the presence of carbonic acid in association with salts of lime and albumen. Similar views have been recently expressed by Woodhead in a paper on Calcification.

It seems probable and possible that these nodes are produced solely by the pouring out of calcific matters by the dentogenetic cells of the pulp, which take on a catagmatic function identical with that of the osteoblasts in the formation of callus. It is probable that this phenomenon occurs in the case of the growth of "pulp

1 "American System of Dentistry," vol. i., p. 862.

nodules" and during calcareous degeneration of the pulp. And if so there, why not here?

That the process must be somewhat of this nature would seem to be confirmed by the interesting case fully reported by C. S. Tomes, and published in the *Transactions Odonto. Soc. of Great Britain*, pp. 184-5-6, 1896. Of such importance this would apparently be, that no excuse is needed to here add Tomes' description. The tooth, a mandibular molar, had been fractured during attempted extraction three years before removal of the root. The pulp cavity was then seen to be occupied by a "cauliflower-shaped mass of shining polished ivory projecting above the original surface of fracture and overflowing on to it." The pulp was only partially calcified, the lowest radicular portion containing sentient tissue. The large mass of newly formed material was closely coherent to, and contiguous with, the old dentine.

Histologically speaking, a cursory inspection of its structure showed a free surface with laminæ running parallel with the surfaces. A series of lacunal spaces came below, then a few dentinal tubes, and finally in the deepest portion, numerous dentinal tubes which were, in some places, continuous with the tubes of the primary or original dentine. The outgrowth of hard tissue contained embedded in it several detached and displaced splinters of the primary dentine.

The following are details:—

"*The Laminated Outer Layer*.—This consists of laminæ parallel with the surface, and varies in thickness reaching in places $\frac{1}{250}$ inch, and containing about ten well-marked layers. Here and there it constitutes the whole of the overflow, and it contains some canaliculi, taking a direction perpendicular to the surface, and a few well-formed lacunæ with their canaliculi. It is present everywhere, though its amount and the distinction of the lamination are variable.

"*The Lacunal and Interglobular Spaces*.—The tissue immediately below the laminated layer is characterised by an immense number of lacunæ and interglobular spaces, which are in parts well formed, and in other parts very coarse and irregular. The fine boundary of this region of lacunal spaces is in places well defined, and terminates with bodies of the "encapsuled lacuna" type; elsewhere it passes insensibly into the region occupied by tubes, in the outer part of which latter region interglobular spaces are abundant, and are somewhat irregularly disposed.

"*The Tube System.*—In the centre of the tooth the tubes, like those of normal dentine, run vertically upwards towards the surface, while towards the sides of the new mass they radiate outwards, passing thus beyond the limits of the fractured primary dentine, and spreading themselves fanlike over the edges of the original tooth to a certain extent. In that portion of the newly formed dentine which lies within the original dentine, and which latter constituted originally the lateral walls of the pulp cavity, the tubes run more or less outwards, and are joined up into continuity with the old dentinal tubes, there being generally an abrupt bend and some dilatation at the junction. From an inspection of the sections it will thus be seen that the whole boundary of the resultant pulp cavity, formed at its sides and below by the original dentine, and above by the new dentine, is formed of dentinal tubes of normal appearance, and that the pulp, though diminished in bulk, has almost perfectly normal surroundings over nearly its whole area.

"As the tubes run outwards they become more widely separated owing to their fanlike spreading: it is noteworthy that there are not a greater number of tubes in the expanded portion, but that the interstices between them become larger. A good many lateral branches are given off, such as those which occur abundantly in the dentine of roots of normal teeth. Towards their outer extremities many of the tubes show longitudinal dilatations, and are joined up to the canaliculi of lacunal spaces; some end in brush-like expansions, while others terminate in loops, the loops being common to two or more tubes; others are sharply bent back on themselves. At and above the ends of the tubes fine globular formations may here and there be very distinctly seen.

" *The Included Splinters of Dentine.*—As has already been mentioned this specimen is probably unique in that the newly formed dentine contains quite a number of little detached pieces of the original dentine of the tooth which were splintered off in the original attempt at extraction, and which have become solidly enclosed in the new formation. They have been displaced in various ways so that their tube systems run in all sorts of directions, and are in no way conformable with the tubes of the new growth. But they have in their irregularity of position, this much in common, that the tubes of the new growth when they are of any size, do not pass beyond them, but terminate beneath them. To this, however, there are some exceptions, where quite small chips appear to have been driven more

deeply into the pulp.   Upon the whole, then, it may be said that the broken fragments of old dentine either lie embedded in the region of lacunal spaces, or between this and the commencement of the tube system.   It is not a little remarkable that none of the fragments show the least sign of absorption, but that their edges are left quite angular, just as if they were broken off.   Where the tubes commence close against the fragments, they are bent about, obviously with relation to the included pieces.

"*Marks of Absorption.*—It is notable that notwithstanding the violent irritation to which the pulp was subjected, in very few places can any marks of absorption be found.   "The occurrence of 'encapsuled lacuna-like' forms has already been mentioned where the lacunal region merges into that of well-formed tubes, but a few marks of absorption and subsequent calcification are to be found elsewhere and in unlikely places.   Thus under the calcified overflow are some pits occupied by Howship's lacunæ."

Here is a history of the injury extending over a period of three years—it is *not* a question of hours or weeks; and so it would appear that the two cases previously quoted might, had time been allowed and all conditions favourable, have succeeded in developing into complete calcification of the pulp, not by the conversion of blood-clot or organisation of the inflammatory products necessarily, but simply by a conversion, or secretion or excretion of the lime-bearing cells of the pulp into one united mass; in other words, the calcification of a plastic exudation.   It is true that no cell of this description has ever been seen depositing this material in the form of calco-globulin; but it is quite easy to understand that as a result of pathological changes in the dentine, these small cells may be stimulated to exercise their functions of abstracting the lime salts from the blood in the pulp, manufacturing them again in the cell-cytoplasm, and ultimately pouring them forth when their work is done.

3

Again, exposure of the pulp through caries leads, as is well known, to inflammation.   This may be so severe as to cause death of the pulp, whose unyielding environment prevents much swelling and the free discharge of accumulated inflammatory products.   Restorative processes are, however, often brought about, and the result is the formation of a so-called "polypus" of the pulp.   The term "polypus" is an incorrect appellation, and as such ought to be deleted from dental terminology (see page 165).

The formation of a protective covering by the extension and growth of squamous epithelial cells on the free surface of the exposed tissue must certainly be considered as another attempt at healing, and might almost be a modification of autoplasty. For chronic inflammation of the pulp, with an epithelial surface, may, and often does, remain in a carious tooth for months and years without any discomfort to the individual. Probably the reparative process is most complete when the new tissue assumes the form not merely of several layers of stratified squamous epithelium, but dips down into the granulations and gives to their surface the shape of simple or compound papillæ. This produces a thicker envelope, and affords greater protection to the soft subjacent structures.

4

Perhaps, after all, the methods of healing of wounds of the pulp are better illustrated in cases of those fractures of the teeth where the parts have been kept *in situ* for some period of time subsequent to the accident. And one may cite, in addition to Tomes' unique specimen, three more cases which have been recently reported.

In the first-mentioned patient—a girl, aged 14—an incisor was fractured longitudinally and impacted. It was thought that union would be established either through the medium of the pulp or periodontal membrane. Here, however, probably from the mobility of the fragments, the pulp became inflamed, fungated, and finally calcified. Thus healing of the pulp lesion occurred, although union of the hard parts was not established.

In a second interesting case several features were noticed. The tooth, also a maxillary incisor, was not impacted, and there was no ultimate union. Under the microscope the margins of the pulp canal showed traces of absorption, Howship's foveolæ being strongly pronounced. These areas of absorption were filled with a layer of new dentine, which at the extreme apex of the root was homogeneous—like the matrix of hyaline cartilage—and cellular, higher up, an intermediate kind of irregular fibrillar dentine intervening. The pulp at the free margin of the upper fragment was enlarged and chronically inflamed, being unusually fibrous in character. How can the presence of these new dentines in the root canal be interpreted? Obviously, a healing process, which, if allowed to continue, would have finally obliterated it with a hard mass of new dentine.

FIGS. 179 and 180.—Outer halves of an impacted united maxillary incisor. The sagittal section, from which Fig. 181 is taken, was the central portion of the tooth. These and the three following photographs are from specimens in the collection of F. J. Bennett.
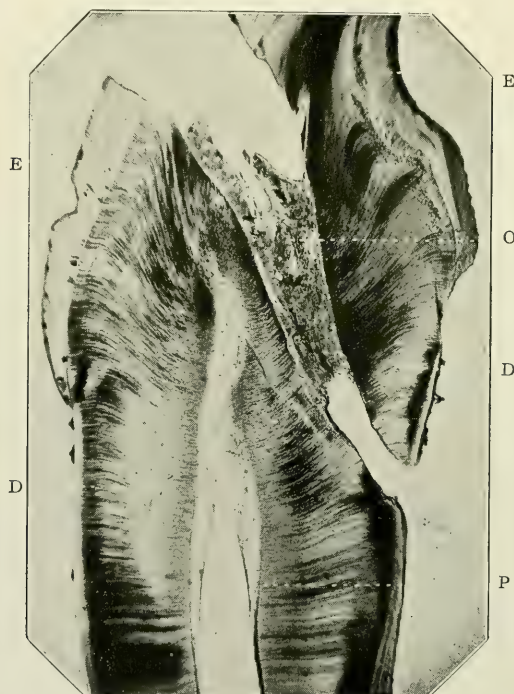
FIG. 181.—Sagittal section, showing the parts. E. Enamel; D. Dentine; P. Pulp cavity; O. New ossified material which acted like a definitive callus.

The case presented somewhat similar features to that of another case of impacted fracture of a canine, save that in the latter the calcified uniting portion was the product, most probably, of the cells of both pulp and periodontal membrane, a genuine example of autoplasty.

This specimen was one of profound interest.

The history showed that an oblique fracture of a tooth and impaction of one of its fragments had remained *in situ* for ten months. Intense pain led to its ultimate removal from the mouth.
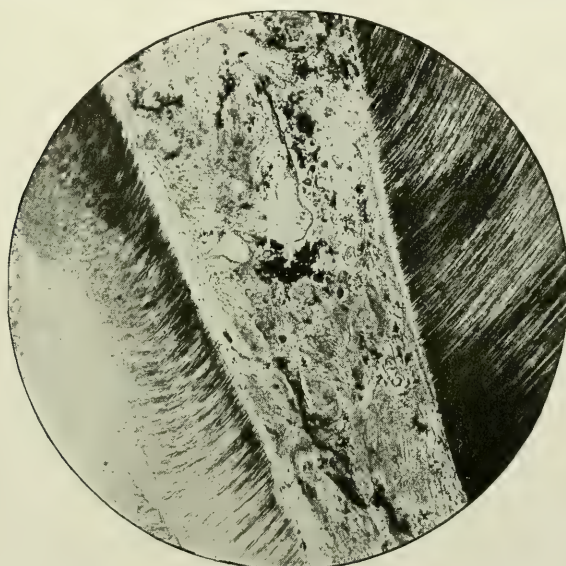
FIG. 182.—Low power magnification of the intervening uniting tissue.

The fragments were united by some dense calcified material which spans the intervening space (see Figs. 181, 182 and 183). The margins of the space were uncalcified. The catagmatic material consisted of spongy or cancellous osseous substance, freely supplied with blood-vessels. The edges of the primary dentine showed signs of absorption and subsequent deposition of a cementum-like material.

The probable explanation of the pathological changes which produced such an excellent union may be described as follows:—It is "a case in which, hæmorrhage having taken place, a natural capping of the exposed pulp occurred, somewhat similarly to the way

a wound heals under a scab. Blood was poured out between the
fragments, organisation took place, numerous blood-vessels were
produced, and ultimately calcification occurred; and, eventually, if
it had been left long enough, the whole of the space would probably
have been filled up with calcified material more or less resembling
bone, or bone and cementum together" (*Trans. Odonto. Soc. of
Great Britain, April*, 1896).

Finally (5) little need be said here with regard to the last di-
vision of this subject, viz., those cases of non-exposure of the pulp,

FIG. 183.—Higher power of the same.

because the question has been discussed in the preceding Chapter,
and, it may be added, is still incompletely investigated. Suffice it
to draw attention to the fact that the inroads made by caries in the
dentine and enamel is counter-balanced in the pulp by the produc-
tion of several different kinds of adventitious dentine, and that
both early and later lesions of the hard parts are nearly constantly
associated with the development of new dentines, and, therefore,
with one of the most interesting methods of repair of the tissues
of the pulp.

CHAPTER VII

THE DEGENERATIONS OF THE DENTAL PULP

Microscopical Elements in:—(i) Fibroid; (ii) Atrophic; (iii) Fatty, and
(iv) Calcareous Degenerations.

GENERAL CHARACTERISTICS

As a result of various conditions, the pulp is exceedingly liable to
undergo degenerative changes. The most common is calcareous,
because of the great tendency for this organ to repair any breach of
its surface, which has been made by carious or other morbid processes.

If a tooth has persisted for years, and has withstood all attacks of
a traumatic, chemical or carious origin, its pulp will, under favourable
circumstances become sclerosed and fibrillar, if not, it will probably
undergo calcareous or other atrophic changes. It is necessary to
consider these conditions somewhat in detail:—

1. Fibroid degeneration.
2. Atrophic degeneration.
3. Fatty degeneration.
4. Calcareous degeneration.

I

Fibroid Degeneration

Whilst studying the histology and histo-pathology of the dental
pulp in its normal relationship to dentine, and preparing sections of
human teeth with this object in view, the author found among his
sections some excellent and remarkable examples of fibrosis or fibroid
degeneration. The typical appearances presented on microscopic
examination of these specimens warranted more than a passing notice:
and as they were further investigated, they became more and more
interesting, sufficiently justifying an accurate description and a care-
ful account being placed on record.

Definition.—Later it will be clearly shown that the term "fibrosis"
or fibroid degeneration is the only one which can with certainty be

applied to this particular form under notice. That it is an example
of degeneration there is no doubt, and it is equally easy to eliminate
those other degenerative varieties—such as mucous, calcareous or
fatty, which animal tissues may undergo.

FIG. 184.—Vertical section of fibroid degeneration of the pulp *in situ*. Pre-
pared by the Author's process. Stained with acid rubine. Magnified 45 times.
D. Dentine; P. Pulp tissue; S. Circular spaces cut longitudinally; F. Dense strands
of strong coarse fibres.

Etiology.—The present instance affords an opportunity of ex-
amining certain structural metamorphoses in the pulp which are
believed not to be dependent on any inflammatory condition, but
simply attendant on and produced by senile constitutional changes.
Further investigations will go to prove that it is a natural old-age
termination of the life of a healthy pulp.
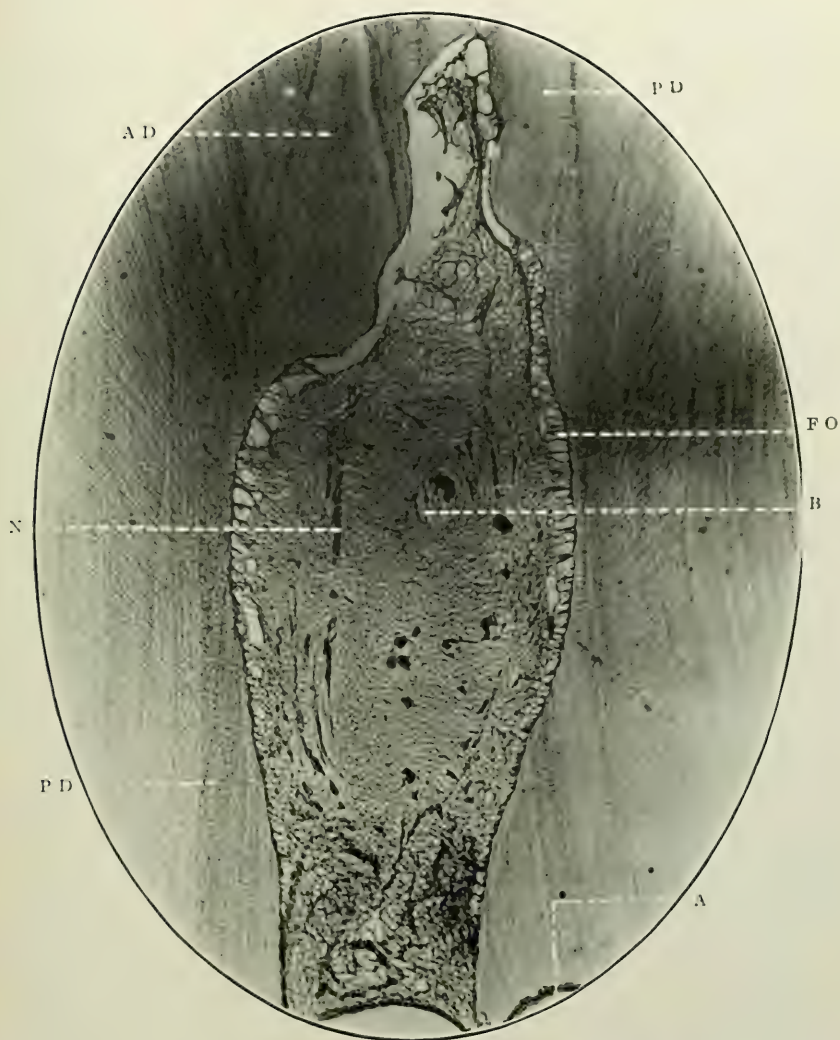
FIG. 185.—Fibroid degeneration of the pulp of a deciduous tooth. Stained with hæmatoxylene. Magnified 45 times. P.D. Primary dentine; A.D. Hyaline adventitious dentine; A. Absorption of primary dentine; F.O. Fibroid odontoblasts; B. Blood-vessel; N. Nerve bundle. The degeneration is complete at the lower part of the figure.

This affection seems to have been unknown to, or overlooked by the dental pathologists of the Continent and America, for though Arkövy, Magitot, Rothmann and Black, publish minute descriptions of the patho-histology of the dental pulp and periosteum, and elaborate dissertations on ulcers and tumours connected therewith, in no instance does one find an account of the senile variations in the tissues of a tooth. Black, in the "American System of Dental Surgery" (Vol. i., p. 859), figures and describes an areolation of the pulp, which at first sight resembles fibrification of that tissue, but differs very materially from it in the fact that numerous cells and
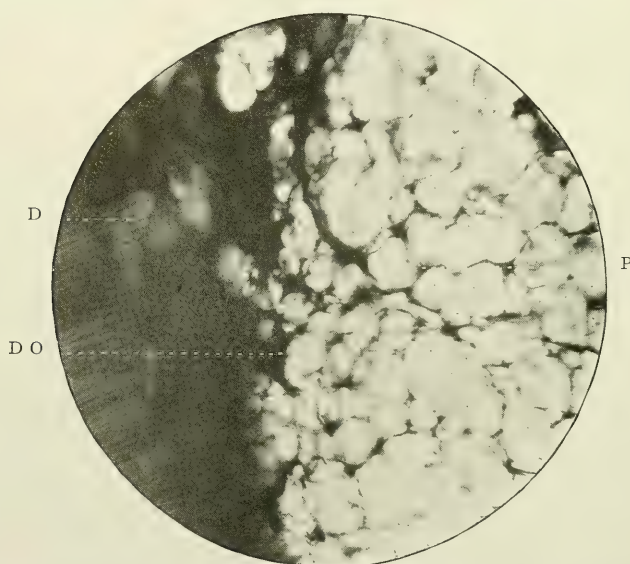
FIG. 186.—Fibroid degeneration of the pulp. Prepared and stained as in the preceding figure. Magnified 250 times. D. Dentine; D.O. Degenerate odontoblasts; P. Pulp tissue proper.

nuclei are present in the sections from which he draws his conclusions. He writes:—"Areolæ develop in the matrix, and all the histological characters of the tissues are profoundly changed. These areolæ were evidently filled with fluid; hence a kind of œdema of the organ must have existed, which, in the enclosed pulp-chamber, has probably gradually destroyed the cellular elements; and new elements thrown out in the inflammatory process, have suffered the same fate."

Of the pathology and clinical histories of the teeth, the histology

of which is presently to be described, little need be said.   The teeth
from which the sections were taken were chiefly maxillary canines
and premolars, apparently sound, but useless, inasmuch as they had
been loosened by the gradual absorption of their alveolar bone,
which had left the portion below the neck exposed.   As a result,
there had occurred that peculiar periostitic pain, or something
analogous, so often noticed in elderly patients with absorbed sockets,
and the teeth were extracted.   They occurred in the mouths of
different patients.   Of a dark yellow colour, they presented, in
addition, all the appearances of the changes of senility.

Similar conditions in the deciduous teeth of children have been
noticed.

A good number of longitudinal and transverse sections, suitable
for microscopic examinations, were obtained.   The revelations af-
forded by these sections are of a particularly interesting nature.

Secondary Changes.—None.

HISTOLOGY

It is evident that in complete pulpar fibrosis no cellular elements of
any description whatever occur.   This is clear at once, and is an
important fact.   No trace of cellular organisation, no vestige of
cell nuclei, no remains of interstitial cement substance can be found
anywhere.   Nerves, cells, blood-vessels, odontoblasts, have alike
shared the process of fibrification, and are no longer recognisable,
and the connective tissue, which is but a loose mass of network in the
normal state, has either become grossly hypertrophied or quite oblit-
erated, and its place taken by a new, firm, fibrous structure, devoid
of cells, nuclei, or any regular arrangement of constituent parts.

In a longitudinal section which is viewed under low magnifica-
tion, the appearances much resemble pulmonary tissue, minus
cells.   The pulp seems to consist of retiform connective tissue,
containing large alveolar spaces, with here and there long cylin-
drical cavities, all having extremely thin walls.   Several of these
tubes are shown in Fig. 184; they are probably the fibrous remains of
what once were blood-vessels.   The dentinal surface of the pulp is
occupied by a more highly defined appearance.   Rows of long, thick
fibres of various shapes and sizes, some bifurcated, others plain, and
again some possessing fibrous off-shoots, are here distinctly seen,
attached to, and in places detached from the dentine.   In those
situations where the fibres adhere, curious dark markings, extending
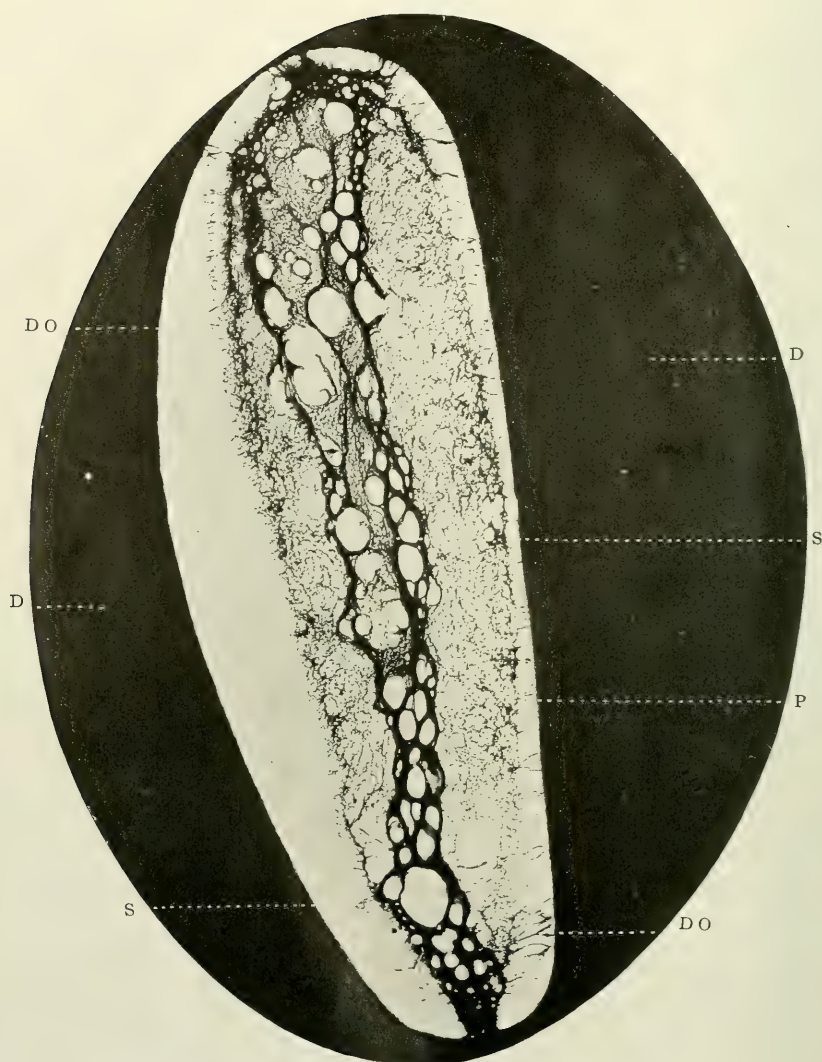
FIG. 187.—Horizontal section of fibroid degeneration of the pulp *in situ*
Prepared and stained as in Fig. 184. Same magnification. D. Deeply stained
dentine; S. Large areolar spaces; D.O. Degenerate odontobasts; P. Fibroid tissue
of the pulp.

in the direction of the lines of the tubules, are visible in the dentine; they are, without doubt, due to the retention, *in situ*, of the dentinal fibrils, which give to them a different refractive index to that of the empty tubes.   These are well demonstrated in the sections from which Figs. 184 and 186 are taken.   The row of fibres represents the pre-existing odontoblasts.   This is proved by the following points:—(1) They distinctly occur at the edge of the pulp, in the site occupied normally by the *membrana eboris;* (2) They are continued into the tubules of the dentine, in a similar manner to that of the processes of the odontoblasts; (3) At the coronal portion of the pulp, the fibres are larger, stronger, more marked, and less numerous than elsewhere; and (4) Their method of attachment to the main parts of the pulp closely approximates to that which obtains in the normal odontoblasts.   Hence it is that these fibres, with their marked outlines, are degenerate and fibroid odontoblasts, but so altered in shape and size as to appear to be merely bundles of connective tissue, which penetrate the dentine to a variable extent.

On examining transverse sections, several curious formations are noted.   First, it is observed that there may be considerable fibroid shrinkage of the pulp, and separation from one side of the pulp cavity, as in Fig. 187, or not so much atrophy as in Fig. 188.   In the first instance, it is perfectly obvious that this shrinkage has not been caused by the mode of preparation to which the tooth was subjected; it is a natural and fibroid contraction of the pulp, produced by a gradual knitting together of the fibrous tissues, which have become on one side detached entirely from the hard dentinal wall, in consequence of the unyielding nature of the latter.   A large conspicuous chain of areolar spaces is, however, the most striking object here observed (Fig. 187).   It stretches without break, across the pulp chamber from side to side, and consists of groups of more or less circular empty spaces, bound together thickly by fibrous tissue. The vacuoles vary greatly in size: the largest measures about 220μ in its greatest length, and 160μ in its greatest width, whilst the smallest here seen, which is also almost circular in shape, measures from 5–10μ in diameter.   At first sight these appear to be blood-vessels cut transversely, but such is not so, as they do not possess their distinctively characteristic walls, neither are they of the same or even approximate diameters.   They are, therefore, simply long cylinders with thin but tenacious boundaries.   No attempts at calcification can be distinguished in them anywhere; the stroma in

13

which they are held is very dense, has a clear fibrous structure, becomes very marked in staining, and consequently is highly differentiated from the surrounding tissue.

It is probably extreme cases only that exhibit so remarkably the chain of areolæ. Earlier stages seem to indicate that the fibrosis originally began in the central portions of the pulp, in the vicinity of the arterial and nervous systems, and that the region of the basal layer of Weil and the odontoblasts were the last to undergo the metamorphosis, as the cells at the periphery of the pulp are the
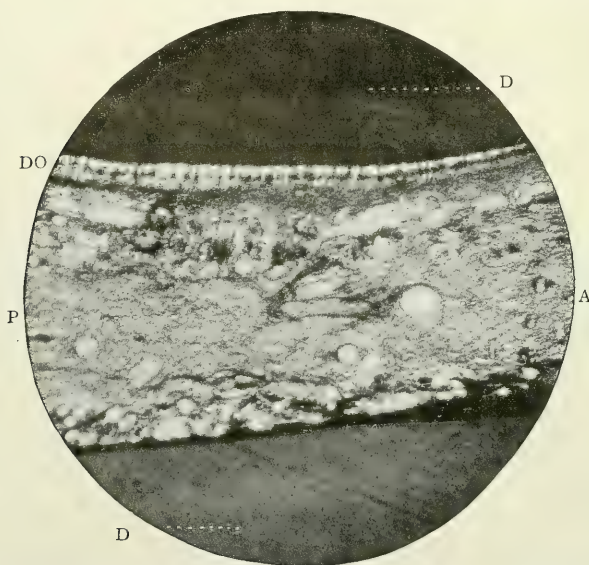
FIG. 188.—Fibroid degeneration of the pulp. A slightly earlier stage than the preceding. Prepared similarly. Stained with Ehrlich's acid hæmatoxylene. Magnified 15 times. D. Dentine; D.O. Degenerate odontoblasts; A. Circular areolar space; P. Fibroid pulp containing no cells, no nuclei, no nerve bundles nor blood-vessels.

last to retain their shape and nuclei (see Fig. 188, which represents an incompleted stage of this form of degeneration).

Degenerate odontoblasts are always clearly visible on the edge of the pulp.

It is worthy of note that the lumina of the dentinal tubules are in no way narrowed or occluded by any adventitious varieties of calcification.

It must not be imagined that these conditions are to be demonstrated in every senile tooth. Dentine nodules, atrophy, total

disorganization, and a number of other pathological states may be, and are often met with: there must be many aberrations from the types here considered. But, given teeth which have been healthily preserved through all the vicissitudes of life, unattacked by extrinsic, and unexposed to intrinsic influences, it would seem that the above results are one form of the natural and usual termination of the life-history of the dental pulp.

The causes of this condition are discussed in Chapter IX (*q. v.*).

2

Atrophy of the pulp

This rare condition has been described and figured by Wedl.[1] It is interesting from a microscopical point of view, but clinically it is indistinguishable from other senile changes.

HISTOLOGY

Here a reticulum fills the pulp cavity; the odontoblasts in early stages are shrunken; if the atrophy has far advanced they will have disappeared. The capillaries freely anastomose, and present, in places, marked varicosities due to the contraction of the connective tissue stroma. They are larger than usual, and have thin walls. No nuclei in their sheaths can be seen.

The nerve sheaths are fatty, granular, and in places deposits of lime salts cover them. The tissue in parts is naturally stained. This is due to escape of the colouring matters of the blood. Rounded, elliptical, or cylindrical deposits of dentine frequently exist in the body of the pulp tissue.

Otto Walkhoff, in describing the photomicrographs of atrophy of the pulp in his "Atlas of the Pathological Histology of the Human Teeth," says that the condition may be limited to the odontoblast layer, vacuoles being found in the midst of the normal healthy tissue. At the same time, the underlying pulp tissue has become condensed and permeated with cells, the vessels are considerably dilated, and presumably indicate the extent of the alterations which the organ has experienced. Frequently the odontoblasts fuse into sheaves or layers, so that they can no longer be individualized (see Fig. 212). The pulp tissue proper exhibits numerous globular spaces, and there is accompanying reticular atrophy.

[1] "Atlas zur Pathologie der Zähne," Plate IV. Leipzig, 1869.

And Armin Rothmann (*op. cit.*) describes a sclerosed atrophy of the pulp ("*Atrophia pulpæ scleroticans*") which is apparently a stage, like the preceding, proceeding to its complete fibrosis. Thus, the normal connective tissue fibres—fine, delicate, and interlacing—have become coarsely fibrillated, and a simultaneous diminution in the number of the cellular elements has occurred. The fibres arrange themselves in narrow layers, which surround the calcific deposits in the sclerosed connective tissue. As the minute calcareous concretions increase in size, through fresh peripheral deposition, so do they form confluent masses of a bright granular material.

3

Fatty Degeneration of the Pulp

Etiology.—This is said to be found in senile teeth, deciduous teeth which are undergoing absorption, and in teeth the pulps of which have been "capped."

Macroscopical Appearances.—To the naked eye the pulp appears to have diminished in size, receded from the margins of the cavity, and of a pale reddish-gray colour.

HISTOLOGY

Fat globules are observed, when examined by the microscope, following the course of the capillaries and nerves, whose walls and sheaths undergo degenerative lipogenesis.

The odontoblasts are degenerated, and have become mucoid or fatty.

4

Calcareous Degenerations of the Pulp

These are very frequent accompaniments of inflammation of this organ as has already been seen; but they frequently exist also in apparently sound teeth unaffected by caries. It is generally believed that a deposition of calcific material is found in the pulps of elderly people, and, as such, is Secondary Dentine. But it is often found in early adult life, and in youth, in sound teeth extracted for regulation purposes. The degenerations have been divided into two classes: 1st, in which the new material is unattached to the walls of the pulp cavity, and 2nd, where it is attached.

1st.—The unattached deposit of the lime salts, a condition which
may be known as calcification of the pulp, may take the form of
nodules or rods.   Nodules are said to be common in the teeth of
gouty patients, in senile teeth affected by fatty degeneration or
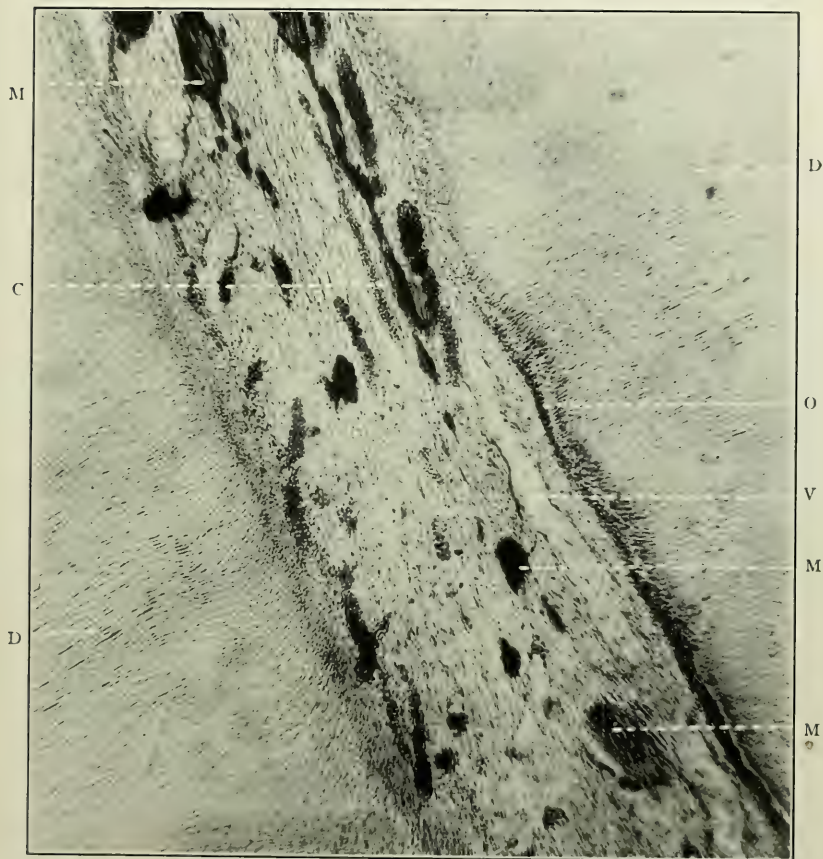
FIG. 189.—Longitudinal section through the pulp of a deciduous molar show-
ing calcareous degeneration.   Stained with hæmatoxylene.   Magnified 45 times.
D. Dentine; O. Odontoblasts; V. Venule; C. Capillary filled with erythrocytes;
M. Masses of calcific material.

reticular atrophy, and in teeth which have been for a lengthy period
subject to attrition or abrasion.   They may exist in apparently
healthy teeth, in which cases, their exact origin is extremely doubtful.
A pulp nodule originates in the centre of the pulp, close to the
vascular system (Figs. 190 and 192).

HISTOLOGY

Its true structure is revealed by the aid of the microscope. Under low powers, Figs. 190 and 191, one observes small, solid, rounded, highly stained masses occupying the centre of the tissue, placed between the vascular and nervous systems. They inconveniently crowd on the nerve bundles, and if allowed to increase by fresh external depositions, will gradually cause a mechanical lateral pressure on these nerve bundles and induce pain. Under higher magnifications the nodules are seen to be made up of matrix and
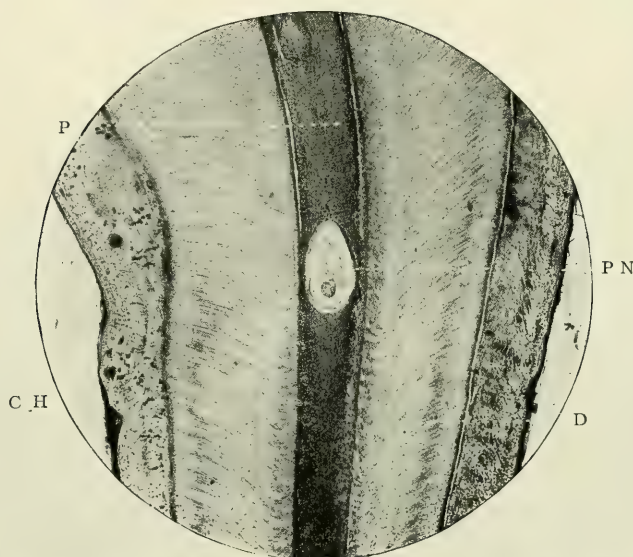
FIG. 190.—A small pulp nodule in the radicular region of the pulp. Magnified 15 times. P. Pulp *in situ;* P.N. Pulp nodule; D. Dentine; H.C. Hyperplasic cementum. The section was prepared by Sidney Spokes.

cells. The former is practically homogeneous in many sections, but it may be laminated or have a distinctly fibrous structure. It is generally rounded or lobulated in outline, and has ragged edges in sections prepared by the author's method. Nodules which have been isolated from the pulp have usually smooth outlines (Fig. 195).

The cellular elements of these nodules are interesting. Roughly speaking, two varieties of cells are discovered, round, nucleated small cells, and long fusiform or spindle-shaped cells, with small nuclei. These, intimately mixed together, are distributed throughout the mass, but at the edges they are chiefly noticeable, giving a
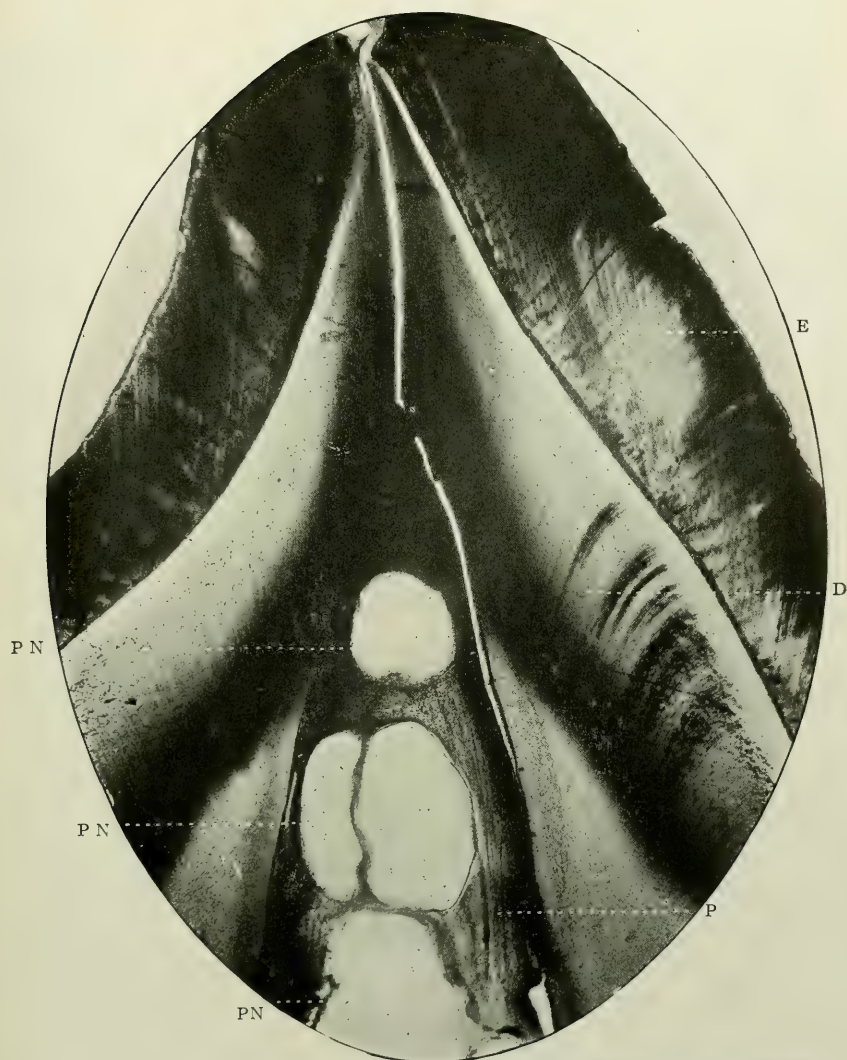
FIG. 191.—Sagittal section through a human permanent canine tooth removed on account of mal-position. From a young patient. Prepared by Weil's process. Magnified 45 times. Shows four pulp nodules *in situ*. E. Enamel; D. Dentine; P. Pulp tissue; P.N. Nearly structureless pulp nodules. Note.— The specimen had remained in Grenacher's alcholic borax-carmine for a period of several years, with the result that the fine tubes in the nodules were stained by long immersion in the colouring reagent.
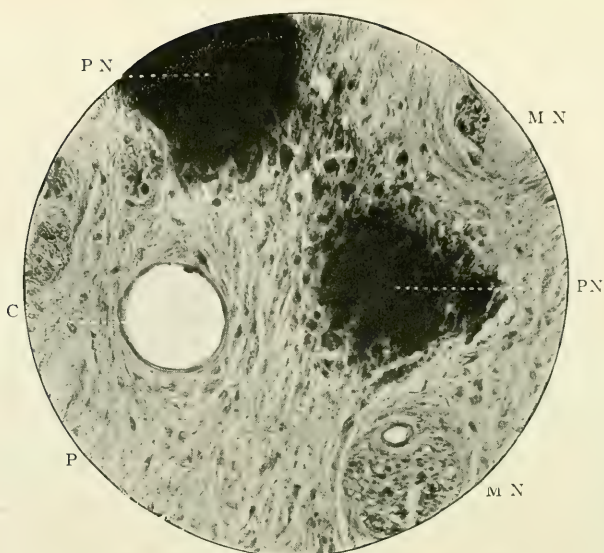
FIG. 192.—The formation of the pulp nodules. Prepared by the Author's process. Magnified 230 times. P.N. Pulp nodules; M.N. Myelinic nerve bundles; P. Pulp tissue; C. Capillary.
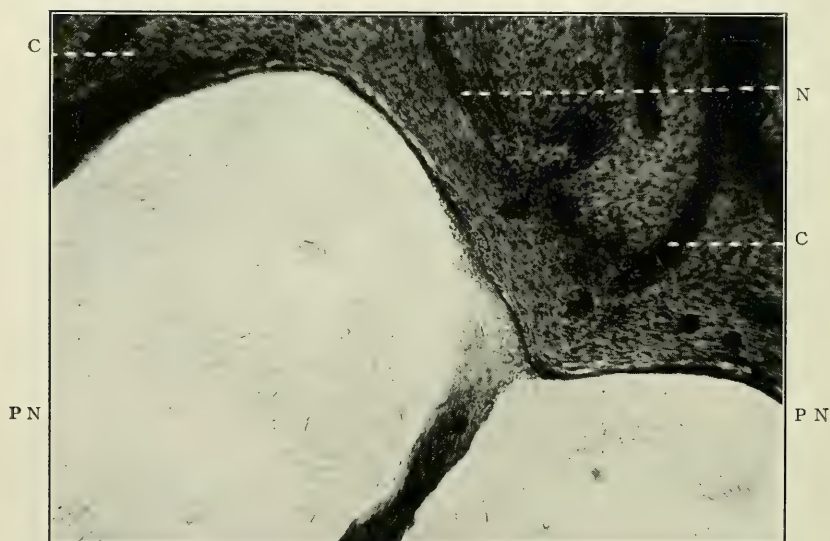
FIG. 193.—Longitudinal section of a pulp containing two large pulp nodules, parts of which are shown. Stained with hæmatoxylene. Magnified 45 times. P.N. Nodules showing striations; C. Capillary; N. Myelinic nerve fibres.

rough uneven outline to the nodule (Figs. 192 and 194).   The presence of the cells in the interior of the nodule is not constant.

Having studied these depositions of lime-salts under many conditions, and possessing sections which exhibit various stages in their development and growth, the author has come to the conclusion that they are formed by a secretion or conversion of the small round cells of the pulp (Fig. 194).   This process may end in the total obliteration of the cell-wall and nucleus, or the cell itself may persist *in situ*.   In some sections the odontoblasts have been
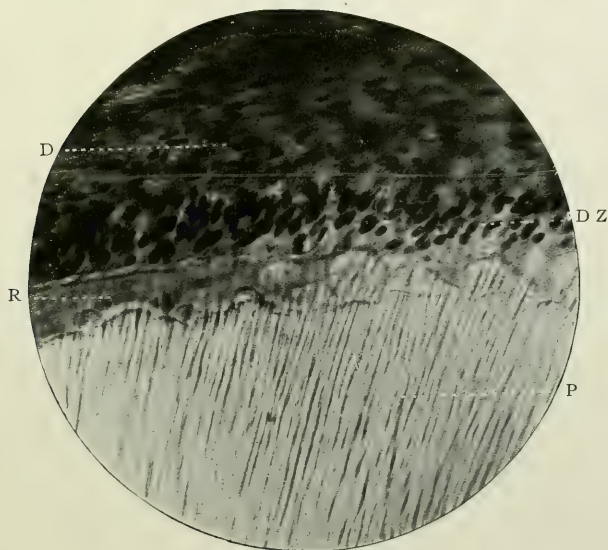
FIG. 194.—The method of construction of the dentine of a pulp nodule.   Prepared by Weil's process.   Magnified 250 times.   D. Formed and calcified dentine; D.Z. Dentogenetic zone; R. Round cells forming the dentogenetic zone; P. Pulp tissue.

caught and embedded in the new dentine when it is attached to the primary or first-formed dentine.   Of course, in the centre of the pulp, odontoblasts are non-existent, and consequently are never found embedded in the mass.   Moreover, they are never seen round its margins.

The fusiform cells are entangled in the secretion which seems to have flowed round them.   They, too, undergo obliteration. Sometimes these completed pulp nodules remind one of the calcospherite spherules found occasionally in the normal tissue of the

periodontal membranes of young teeth. When quite complete, they may be more or less laminated, may be quite structureless, or may have a few fine tubes radiating from the centre outwards. These tiny tubes have been probably produced by the long fusiform cells.

It has just been stated that pulp nodules are invariably solid. This is not, however, quite correct.

The accompanying photomicrograph (Fig. 196) shows that sometimes a pulp nodule may contain a pulp chamber in its interior. An example of this occurred recently in the practice of Mr. Neville
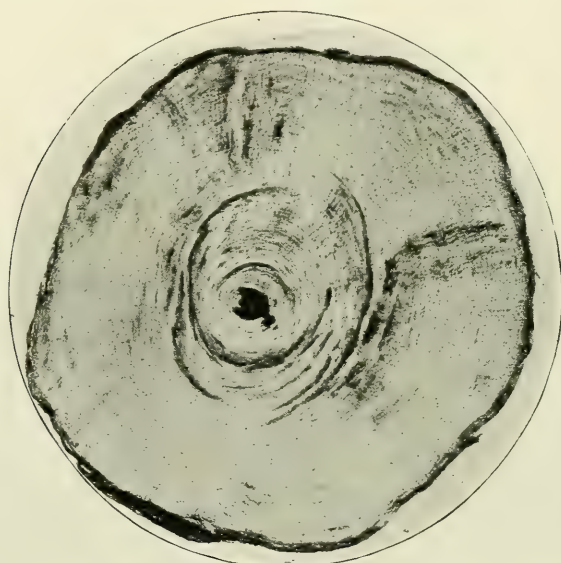
Fig. 195.—A pulp nodule isolated from the pulp. Shows its central nuclear formation and its concentric lamination. Prepared by grinding. Magnified 50 times. (*From the collection of G. W. Watson.*)

Davis. Three teeth were removed from the same mouth, on account of excruciating and incurable pain. The pulps contained the largest nodules probably on record. One of them measured in width 2.5 mm. and in length 10 mm. Extending in the central axis of the nodule is a canal filled with ordinary pulp tissue. In one section the outermost part of the nodule consisted of fine-tubed orthodentine which gave the growth a conical shape. Inside this and filling up what at one time must have been the pulp cavity, was a mass of calcareous material, with still a small amount of
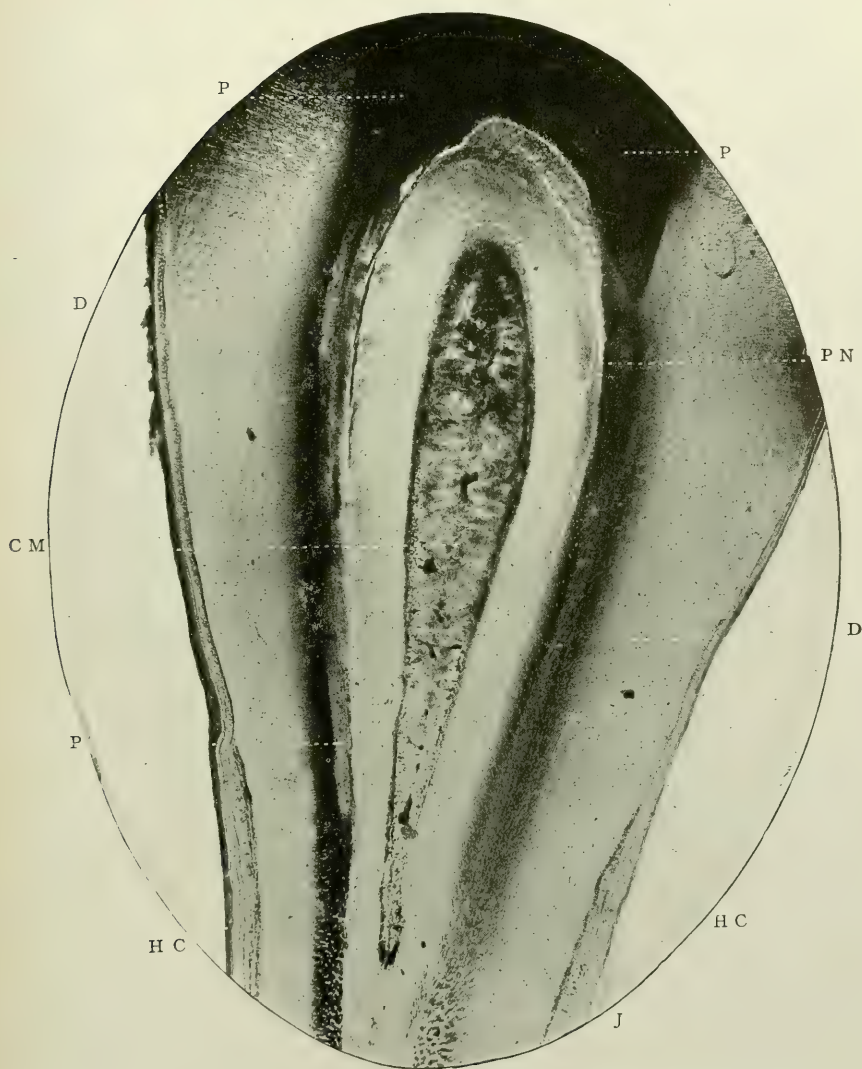
FIG. 196.—A pulp nodule *in situ*, containing an axial cavity filled with a calcified mass resembling hyperplasic cementum or compact bone. Prepared by Weil's process. Magnified 12 times. P.N. Pulp nodule; C.M. Calcified material occupying its interior; P. Pulp tissue; D. Dentine; H.C. Hyperplasic cementum; J. Point of junction of pulp nodule with the primary dentine of the tooth.

pulp tissue remaining.   The central hard mass contained a granular matrix, in which were embedded great numbers of lacunæ and inter-globular-like spaces.   Here and there a few scattered dentinal tubes radiating centripetally were arranged in bundles.   Such cases have
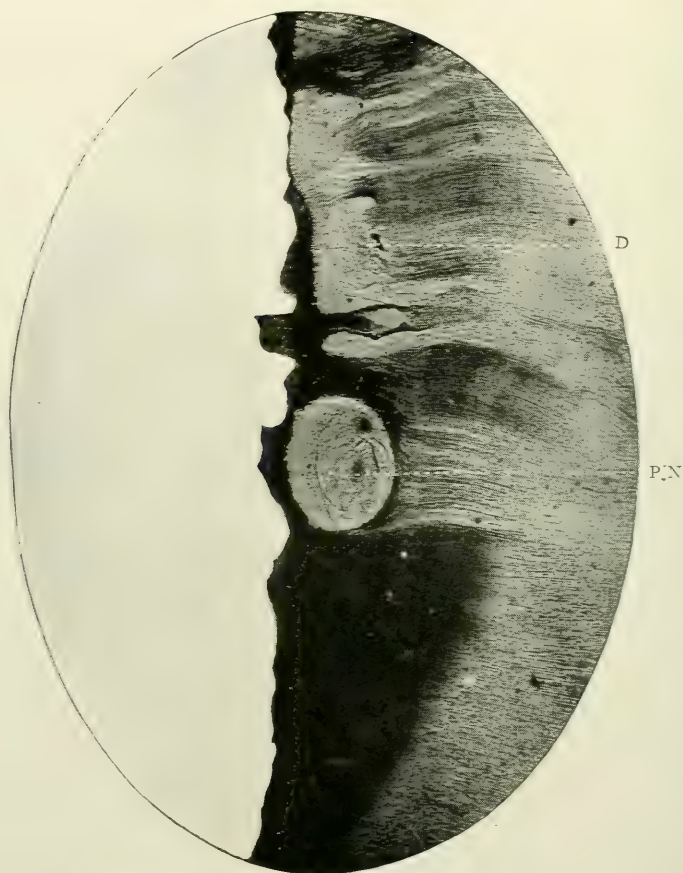
FIG. 197.—A pulp nodule fused to the parieties of a pulp cavity.   Prepared by grinding.   Magnified 15 times.   P.N. Pulp nodule; D. Dentine of the tooth. (*The section was lent by J. F. Colyer.*)

never been described before; but they are probably not unique. (Fig. 196).

One or more nodules may exist in the same pulp.   They are oftenest located in the coronal or cervical regions, but occasionally may be found, as in Fig. 190, near the apical portion of the root canal.

Occasionally a pulp nodule may be attached to the wall of the pulp cavity. This has been brought about by new dentine from the margin of the pulp cavity, increasing and gradually encircling the nodule.

2nd.—*Attached deposits* of dentine are generally seen in the coronal or cervical portions of the pulp (Fig. 197). They are associated at times with its hyperæmia and inflammation or, at times, any condition, like erosion, caries, etc., where there have been pathological changes of the dentine.

Where they are due to the latter and there is a breach of surface, they represent the condition named by Salter, "dentine of repair."

Generally speaking, these new masses of dentine include many varieties. Thus they may differ but little from normal fine-tubed dentine—the tubes perhaps being finer and less evenly distributed. They may be almost structureless; again they may contain a few nucleated cells, or spaces which closely resemble interglobular spaces.

In aged teeth, the pulp may become partially or completely calcified. Long rods of calcific material run longitudinally through its substance, and, in time, fuse and ultimately obliterate all traces of soft tissue (see Fig. 89).

CHAPTER VIII

THE PATHOLOGY OF THE PULP IN RELATION TO CLINICAL DENTAL SURGERY

Introductory—Carious lesions—Lesions due to Tactile, Thermal, Chemical and Electrical stimulations—Referred Pain and Obscure Reflex acts—Receptivity of the Pulp: Its Hyperæsthesia and Dysæthesia—Phases of Degeneration.

INTRODUCTORY

The above subject is of vast importance to the Dental Surgeon, but unfortunately at present only partially developed. The reason why it is only partially developed lies chiefly in the fact that the microscopical technique associated with its study is so tedious to accomplish, and sometimes so uncertain in its results, that it does not appeal generally to the worker in dental histology in spite of there being ample clinical material and ample scope for research. No thoughtful reader can for an instant deny the immense importance to the dental surgeon which attaches to the subject, especially to one who has lofty ethical ideals and who practices his art in its most conservative forms. All the systematic knowledge of diseases of other organs of the body which we possess—except, perhaps, those relating to myology and osteology—the morbid conditions of the blood-vessels, of the nervous mechanism, of the cellular system, wholly or in part, simple or complicated as they sometimes are, focus themselves on that minute structure in a tooth which we call the pulp, which is physiologically and in very truth the marrow of the tooth.

THE DENTAL PULP ANALOGOUS TO BONE MARROW—A PERFECT ORGAN

There is not so much known about pathological conditions of the marrow of bones generally, but one probably does understand in a measure some changes which the marrow of a tooth has undergone when subjected to local or constitutional disturbances. And this is not at all surprising when one begins to realize that it is a perfect

organ in a way, with its small arteries, veins, capillaries, and myelinic and amyelinic nerve fibres and cells whose functions are partly odontoblastic in nature, and partly are closely associated with the sensory nervous apparatus—a perfect organ situated on the terminal fringe of that complicated tangle of sentient fibres, the largest cranial nerve, the *Trigeminus*. It is on this fact and on all that it implies that the importance of the pulp depends, and that makes its conservation above any other dental tissue of the supremest moment. It would be futile on the part of the author to marshal in review the pathological conditions of which pulp may be a victim. These can be found in preceding chapters. This chapter, although it does not advance any novel or startling hypothesis, is designed to be suggestive rather than assertive, argumentative rather than dictatorial. In other words, *de donner à penser à quelqu'un*, to indicate possibilities, and to chronicle one or two sidelights which seem to have some bearing on the pathology of the organ.

The subject may have been approached in two ways. On the one hand, it may have been considered strictly from the point of view of the association of certain signs, and subjective and objective symptoms and treatment, with the morbid conditions of the pulp; or on the other hand, the diseases and degenerations of this organ might have been systematically and histologically described, and linked in thought with certain clinical aspects of dental surgery.

(I.) Carious Lesions

DIFFICULTIES IN ELUCIDATING CERTAIN PROBLEMS

The author wishes it had been possible for him to have stated clearly and satisfactorily what happens to the pulp and surrounding parts when obtundent drugs have been placed in a carious cavity; what takes place when antiseptic solutions are used, what physical, chemical, physiological, or pathological changes are induced when the various filling materials have been, for a given length of time, brought into contact with enamel and dentine.

A perusal of the papers of Miller and Truman which appeared in the *Dental Cosmos* for 1890 and 1895 respectively, and a search through subsequent literature, serves to show the incompleteness of our knowledge of perhaps the most important and most common condition of things, viz., the exact results which occur in the daily mechanical and chemical treatment of diseases of the vitalized dental tissues and of the effects of dead bodies on living substances.

The meeting-point of the dead and living is indeed a mystery.
It is, of course, admitted that many facts are known about these
things.   James Truman published in the *Dental Cosmos* for January
1895 a most interesting account of his work "On the Relative
Penetrating Power of Coagulants."   In spite of his writings being
punctuated here and there by such words as "possibly," "perhaps,"
"probably," he would seem to have definitely proved that zinc
chloride should not be used as an obtundent of sensitive dentine,
because "it is exceedingly dangerous to the life of the pulp," though
he does not precisely describe what causes this danger, and entirely
ignores the histo-pathological side of the question.   He further
writes: "In every instance, silver nitrate has proved deeply pene-
trating and coagulating with rapidity and certainty very nearly equal
to zinc chloride;" but still he adds, "The result has not been entirely
satisfactory."

The nearest approach, however, to a scientific attempt to unravel
these knotty points was that achieved in 1890, by Miller, who,
nevertheless, limited his observations to the comparative value
of antiseptics used in dental surgery, making use of pulps which,
being isolated from the mouth, had lost their natural living surround-
ings.   These, therefore, at present, are unsolved problems, the
explanations given as to the actual phenomena which occur being
most vague.

DIVERGENT OPINIONS ON CERTAIN PROBLEMS

For instance, Black in his "Operative Dentistry," vol. i., p.
193, 1908 in treating of the question of curative effects of fillings
says: "Fillings cure purely and simply by shutting out everything
from contact with dentine. ... ...   No systemic change is
produced as a direct effect."

Again, Inglis, in the last edition of Burchard's excellent "Dental
Pathology and Therapeutics," writes: "A process of eburnation
is set up when progress of caries is delayed, and in some cases
ceases."   What is the "process of eburnation"?   The writer tells
us: "The continued stimulation of the ends of the dentinal fibrillæ
which are exposed in abrasion causes them either to become
hypersensitive or stimulates them to formative activity.   Tubule
material is built upon the inner walls of the tubules, obliterating
their lumen.   This is the so-called tubular consolidation or calci-
fication (eburnation)."

This statement is in entire disagreement with that of Black, *op. cit.* p. 128, who says: "The suggestion has been made that some additions of calcium salts may be made on the walls of the dentinal tubules, narrowing their calibre. This is plausible, but as yet no sufficient series of measurements have been made to determine the facts." And again: "Dentine or enamel once formed is formed for all time, it can never be re-formed, changed, or improved in its character or qualities."

By certain clinical aspects of dental surgery, of which mention was made just now, is meant the palliative and operative treatment of diseases of the pulp. This tissue with an infinite variety of disease—inflammation and its terminations, degenerations, senile changes, and more remarkable reconstructive potentialities, offers but few opportunities to the dental surgeon for the display of his surgical knowledge and principles and his skill. They may be summarised as two only, the problem being "to cap or not to cap," to devitalise or not to devitalise.

THE QUESTION OF CAPPING EXPOSURES

A superficial or deep ulceration or incised or punctured wound cannot be treated; it is difficult to get rid of an infected condition, or restore a functionless pulp to its normal activity, without the complete destruction of the organ. The anatomical peculiarities of the hard and soft parts are antagonistic to these things. If the operation of capping an exposure of the pulp, under the very strictest aseptic precautions is successful, it is probably because only non-liquefying micro-organisms exist in the dentine and in the immediate neighbourhood of the tooth which is being treated.

In this connection it may be recalled that Goadby (*Mycology of the Mouth*, 1903) has discovered only the *Streptococcus pyogenes* (*viridans*) and the *Bacillus necrodentalis*, which are liquefying organisms, in the deep layers of carious dentine.

In 1900, in Paris, the author (*Trans. Third International Dental Congress*) ventured to emphasise the fact that capping was, as a routine method of practice, always a failure. It seems to him, after a riper experience, that generally the operation is not a success on account of the prevalence and presence of these facultative (obligatory) aerobes, which, according to Goadby, are capable of obtaining oxygen from gelatine, which is the end-product of collagen.

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THE PHYSIOLOGICAL RESISTANCE OF THE HEALTHY PULP

But this is not all. There is another factor which is probably
of greater importance than the presence of these micro-organisms;
this is the physiological resistance of the pulp. It would seem that
when the operation of capping is unattended by pain or by death of
the pulp—no matter how long delayed after the actual treatment—
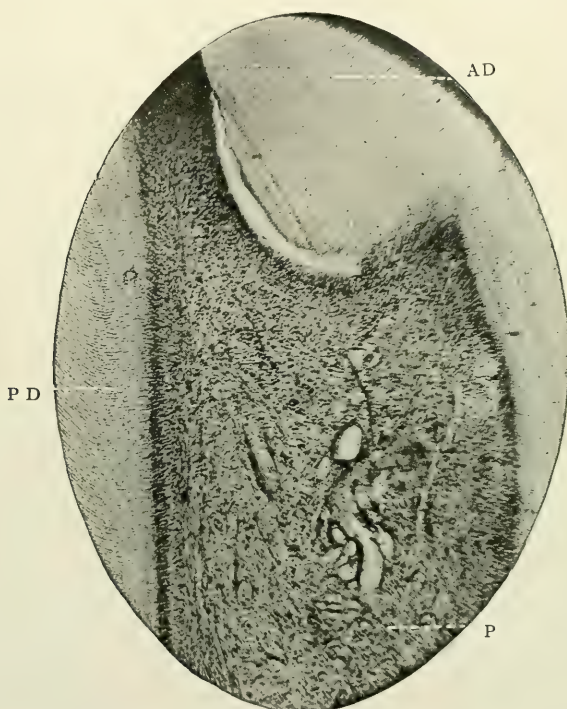
FIG. 198.—Logitudinal · section of canine with pulp *in situ*. Enamel was
hypoplasic, and subsequently, with the dentine, became carious. There are
no micro-organisms in the adventitious dentine. P.D. Primary dentine; P. Pulp;
A.D. Adventitious dentine. Magnified 12 times.

it is due to the physiological resistance not only on the part of the
pulp itself, but also on the part of the adventitious dentine which
has been deposited on its surface. The author has failed in his at-
tempts to stain bacteria in the pulp and adventitious dentine in some
carious teeth which presented a hypoplasic condition of the enamel.
This leads him to suppose that if this adventitious dentine is produced
early by an energetic pulp, and if the caries proceeds at a slow

uniform rate, the pulp will not become infected and the case would be suitable for capping an exposure, if one should by any chance be made, either idiopathically or traumatically or pathologically.

Miller considered that there is in the thin free margin of enamel a certain amount of physiological resistance, as also in the translucent zone of carious dentine.

It is probable, nay certain, that a large amount of physiological resistance resides in the pulp also, as witness the unvarying frequency with which it lays down adventitious dentine. And if a
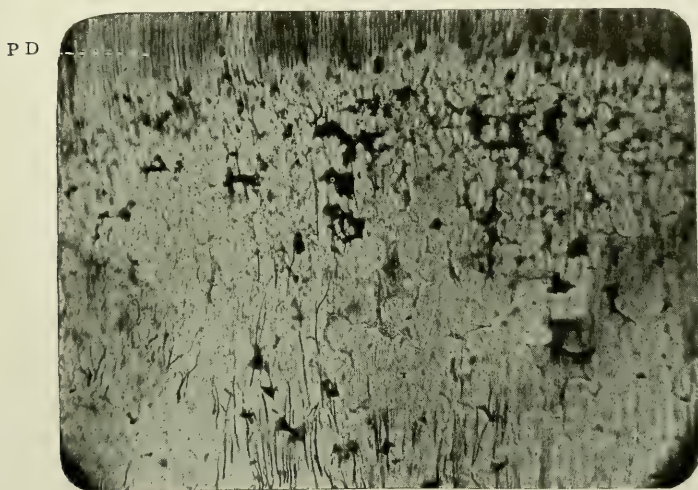

FIG. 199.—Areolar adventitious dentine—a most suitable nidus for the growth and development of the micro-organisms of caries. Rapidly formed as a protection of the pulp. P.D. Primary dentine. Magnified 200 times.

perfect capping has been performed and facultative micro-organisms are absent or few in number, and the pulp always retains its high degree of physiological resistance, the results will be entirely satisfactory.

Therefore, given a healthy pulp, the success of this operation would appear to depend on three conditions: (i) A slight injury, (ii) Absence of facultative aërobes, (iii) Well-maintained physiological resistance of the pulp, and of its adventitious dentine.

Conversely, therefore, anything which destroys, or reduces, or abolishes this physiological reaction, such as prolonged mental anxiety, chronic wasting diseases, often repeated traumatisms of the hard parts, frequent and varied thermal stimulations, will in

the end, in spite of the aseptic care of the operator, tend to bring about disaster, and the pulp will die.

REGIONAL HYPERÆMIA

Dental caries, when acute, is accompanied in its early stages by a regional hyperæmia which according to the part attacked—that is, that which is nearest to the breach of surface—is either coronal, cornual, cervical, or radicular. Sections of teeth extracted
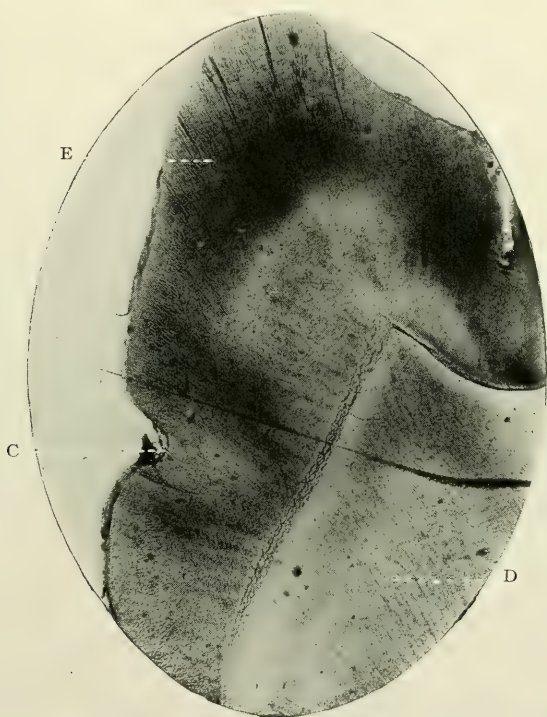
FIG. 200.—Longitudinal section through a cusp of a molar showing signs of "arrested" caries. Surface of enamel and dentine (where exposed) blackish in color. E. Enamel; D. Dentine; C. Caries making breach of surface of enamel prior or subsequent to the general arrest of the process. "White spot" absent; enamel tissue fully stained. Magnified 45 times.

during a paroxysm of odontalgia reveal this quite clearly, but chronic caries does not necessarily produce it. Coronal regional hyperæmia may exist to a slight degree in such caries, but when the physiological resistance of the pulp is fully restored and permanently established, not only does this hyperæmia undergo resolution, but

the contents of the dentinal tubes claim their share in the physiological resistance, and arrested caries results, often the whole phenomena of decay and arrest proceeding without any pain.

ARRESTED CARIES

One would have thought that arrested caries would have been associated with deposits of secondary and adventitious dentine on the pulp surface. This is not necessarily so. Specimens of true arrested caries are not easy to obtain, but those that have been prepared show no sign of this, nor pathological conditions of the pulp. If specimens have been prepared by the Koch-Weil method, even before immersion in the graduated alcoholic solutions, the dentine is extremely hard and resistant to a fine saw, such as an American No. 4. What enamel remains on the surface is also exceedingly dense and difficult to cut. Curiously enough, however, and for what reason it is at present impossible to determine, the enamel rods become stained throughout their length by means of the Grenacher's borax carmine which is used in conjunction with this process (Fig. 200). The transverse striæ are more marked than natural, and the tissue has the appearance of having been washed in a weak acid solution. This staining of the enamel may not occur universally; the calcification of the teeth varies considerably even in the same mouth. Still it is evident that but little clinical significance can lie in this fact, although it is interesting from the histological point of view.

SOME SENILE CHANGES

As age advances, the pulp cavity is not encroached upon to any appreciable extent by new deposits of dentine as a normal event. The author's observations do not accord with Tomes, who remarks in the 1906 edition of "A System of Dental Surgery," page 416, "The area of the pulp cavity becomes gradually diminished by the slow addition of dentine to that which was formed when the tooth was in a state of active growth;" nor with those of Dr. Loos, who, in *Scheff's Handbuch für Zahnheilkunde*, 1908, states that "There must be mentioned a general, physiological, senile new formation of dentine," and continues: "With the entire development of the teeth, normal dentine formation does not stop, but continues slowly up to the greatest period of age. A proof of this is the fact that the pulp

cavity in advanced age is found to be smaller than in earlier years—indeed, it may entirely disappear and this physiological new formation of dentine produces most diffuse uniform thickening of the dentine wall. It appears, however, that this dentine formation does not take place uniformly over the whole wall of the pulp cavity. The root-canals in advanced age are mostly found narrowed; but frequently on the wall of the pulp chamber only certain places are distinctly altered, viz.: those which border on the pulp chamber in a vertical direction. As the size and form of the pulp cavity are sub-
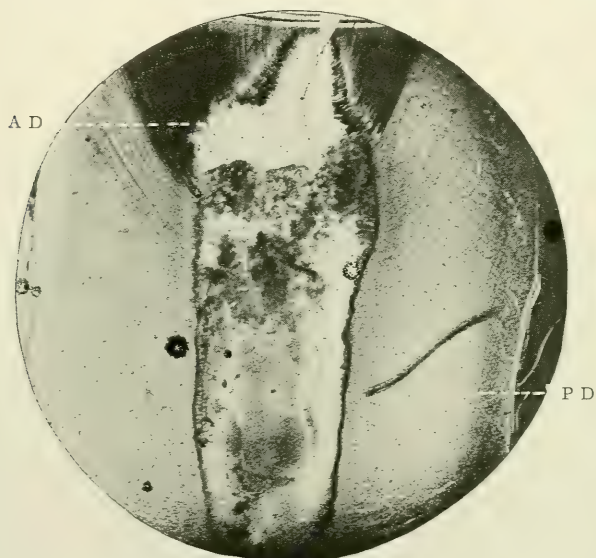

FIG. 201.—Longitudinal section (mesio-distal diameter) of lower incisor of patient aged seventy-one. Pulp chamber filled with débris and detritus by grinding. Small amount of adventitious dentine—marked attrition. P.D. Primary dentine; AD. Adventitious dentine. Magnified 12 times.

ject to manifold individual variations, it is often very difficult to find out whether we have to do with normal or with altered conditions. Histologically this dentine does not exhibit any noteworthy differences from the normal, and it can scarcely be distinguished from substituted dentine or 'odontheles' (pulp nodules)."

If this is a normal physiological process on the part of the pulp, one would expect to find it universally in all senile teeth; but it is not so. The accompanying figure for instance, is a case in point, where the diameter of the pulp chamber measures 3 mm. (Fig. 201). The history of the tooth was as follows: "Male, aged seventy-one.

No inflammation of pulp, slight amount of tartar, tooth loose, not isolated in position, alveolar process absorbed and walls of sockets thinned, 'pyorrhea' present very markedly."

It is quite impossible to understand how calcification of the dentinal tubules can occur and how the pulp chamber can become lessened in size, except by the production of new adventitious dentine. Of course odontoblasts are still present up to the end of the
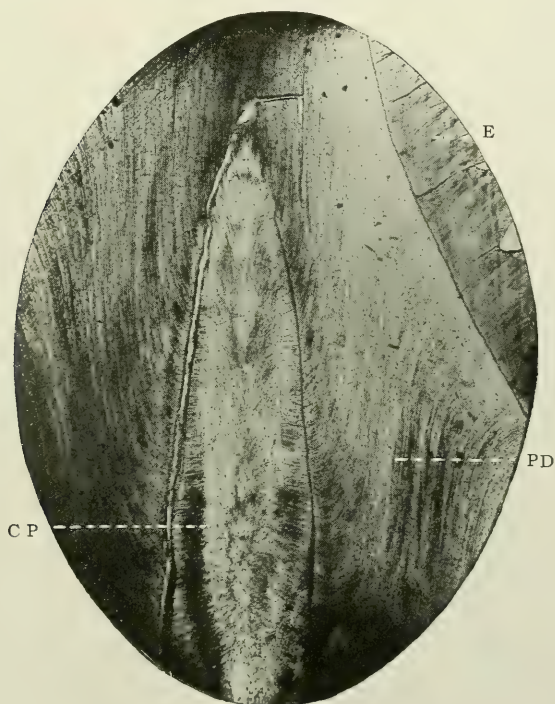
FIG. 202.—Longitudinal section of a senile tooth. E. Enamel; P.D. Primary dentine. C.P. Calcified pulp. Magnified 12 times.

life of the pulp, but they are not functional after the fiftieth or sixtieth year. When the process of dentinification has ceased, and fresh secondary dentine is deposited, the microscope usually exhibits very clearly the line of demarcation between young and old tissue. The idea which has been current for generations probably arose in the attempt to account for the discrepancies in the size of the calibre of the dentinal tubules at their centripetal and centrifugal extremities.

SYSTEMATIC DENTAL HISTORIES

At this juncture the writer must digress slightly from his subject in order to draw attention to the need for and the methods of systematically obtaining reports of teeth which should be of scientific value when research work is being done. He refers to the individual "histories" of teeth. It is important that the investigator should learn thoroughly all he can about the objects he studies, and so it seems imperative that with regard to teeth which are about to be examined the following points should be ascertained: (1) Age of the patient. (2) Sex. (3) Denomination of the tooth. (4) Obvious disease of the pulp or periodontal membrane. (5) Presence of "pyorrhea alveolaris." (6) Position of the tooth or teeth in regard to the dental arch. (7) Conditions of the socket at the time of extraction. (8) Presence or absence of tartar. (9) Presence or absence of any obvious lesions of the hard parts, such as attrition, abrasion, or erosion.

The *Age* of the patient is useful as a general guide as to what to expect. One need not dwell on the importance of this in the young. Apart from the question of age in treating the irregularities in position of the teeth, it is equally essential to ascertain the age of the patient in order to gage somewhat the conditions of the pulp or root membrane. Thus one knows that the apical foramen in the permanent canine is closed shortly after the tooth has erupted.[1] One knows that the pulps of elderly people become less sensitive as years pass by. One knows that there are three cornua to the pulp of the permanent first incisor up to the fifteenth year, when they disappear; and so on. But age does not always prove a reliable guide, for, as has been already shown, very frequently the pulps of children's deciduous and permanent teeth may exhibit senile and other degenerative changes.

Sex.—One cannot recognize with facility the difference, either macroscopically or microscopically, between the teeth of a male or a female patient. Of course this degree of variation is nothing like

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[1] The approximate dates of "closure" of the apical foramina of the permanent teeth may be given as follows: A. The Maxillary series—First incisor, 11th year; Second incisor, 11th year; Canine, 13th year; First premolar, 12th year; Second premolar, 12th year; First molar, 12th year; Second molar, 15th year; Third molar, 19th year. B. The Mandibular series—First incisor, 10th year; Second incisor, 11th year; Canine, 13th year; First premolar, 12th year; Second premolar, 12th year; First molar, 11th year; Second molar, 16th year; Third molar, 21st year.

so great as between the hair, or brain, etc., of the two sexes. The curvatures of the enamel rods are apparently the same; the structure of the two pulps is apparently identical. But the author believes that there is a dissimilarity between them in the size, the structure, and the chemical and functional characteristics of the teeth.

Regarding the first, Amoëdo (*L'Art Dentaire en Médecin Légale*, 1898) records in a tabulated form some measurements made by Mela between the size in transverse diameter of the first and second incisors of man and woman. The figures show that the mean difference between the dimensions in a mesio-distal direction of the first maxillary incisors of the two sexes is 0.627 mm.; of the maxillary second incisors 0.159 mm. The mean measurements between the maximum diameters of the first and second incisors was 2.25 mm. in man, and 1.89 mm. in woman, a difference of 0.38 mm. The maximum diameter of man's first incisors gave a maximum of 11 mm. and a minimum of 7.5 mm., that of the same teeth of woman giving a maximum of 9.8 mm. and a minimum of 7.1 mm. "La différence sexuelle entre les deux extrêmes des incisives centrales supérieures," he writes, "est de 1.3 mm. à l'avantage du sexe masculin. . . . La différence sexuelle entre les deux extrêmes des incisives latérales supérieures est de 5 mm. en faveur du sexe masculin."

Regarding the last, the teeth of women would appear to be less subject to attrition or abrasion; they cannot be influenced by the same amount of muscular power as the former, though the food is more or less identical. The respective dental and muscular dynamics and potentialities of mastication are distinct, and if tested by means of a dynamometer would probably be found to differ.

Comparative anatomy teaches that the teeth of the female vary in development, in size, and sometimes in function, from those of the male; instances need not be given. We also learn that if but a slight amount of work has to be accomplished, tooth structure is profoundly modified. Thus the *Edentata* have no enamel, simply because their food is of so soft a character as to involve practically the abolition of the function of mastication. Again, the enamel of the *Manatee* is arranged in rods which run straight courses; on the other hand, that of the rodents, particularly the beaver, presents a most complicated pattern. The former does not require for eating aquatic plants a dense structure such as the latter, whose teeth are used for gnawing and chiseling the barks and trunks of trees. Hence, at first sight, though it seems strange to suppose this x difference between male and female teeth, there is some support for the idea.

At all events, the author thinks that there is certainly some ground for the belief that the pulps of the teeth of women may and often do undergo unique vascular disturbances at certain periods of their lives; and this fact alone would constitute a fundamental difference between the two.

The *Denomination* of the tooth is important. The enamel of incisors is less complex in pattern than that of the molars. From the chemical point of view, also, there is a remarkable difference. Gassmann, in the *Zeitschrift für Physiologische Chemie*, 1908, discovered as a result of his investigations that human canines contain 29.78 per cent. of calcium salts, third molars 31.65; of water combined with the organic matters, 8.09 in the former and 6.91 in the latter. Pulps appear to be histologically identical, but most probably there is here again a dissimilarity which one is not clever enough at present to detect and acknowledge.

In other words, just as there are no two persons exactly alike, no two animals, no two birds, no two plants, no two blades of grass, so there are no two teeth precisely and in every minute particular the same.

With regard to the other points which have been outlined, which should be given in the histories of cases, one need not dwell on each in detail. One will merely add that a tooth which is isolated in position, from removal of anterior and posterior neighbours, is more likely to undergo morbid changes than one which is part of an unbroken series, owing to the undue strain or shock of mastication; that a loose tooth is a degenerate or degenerating organ, and that the presence or absence of calculus is of great importance, inasmuch as the greater the amount of tartar which incrusts the surfaces, and the longer it remains *in situ*, the less functional the tooth and therefore the more liable to become impaired the pulp.

In addition to this, the naked-eye examination of the specimen must be described, viz.: the amount of tartar (if present); its position on the root or roots; its distance from the cervical margin; its nature; the translucence or otherwise of the apical region of the root; the deflections or normal appearances of the root; the amount of abrasion of the crown, etc.; in short, all the abnormal characteristics of the object under consideration as far as the macroscopical appearances are concerned.

(II.) Non-Carious Lesions

(A) Lesions due to tactile, thermal, chemical, and electrical stimulations

(1) *Tactile impressions* which set up pain are, as is well known, those in relationship with the surfaces of the cervical margins of the teeth. No lesion may exist to the naked eye; but there is a micro-
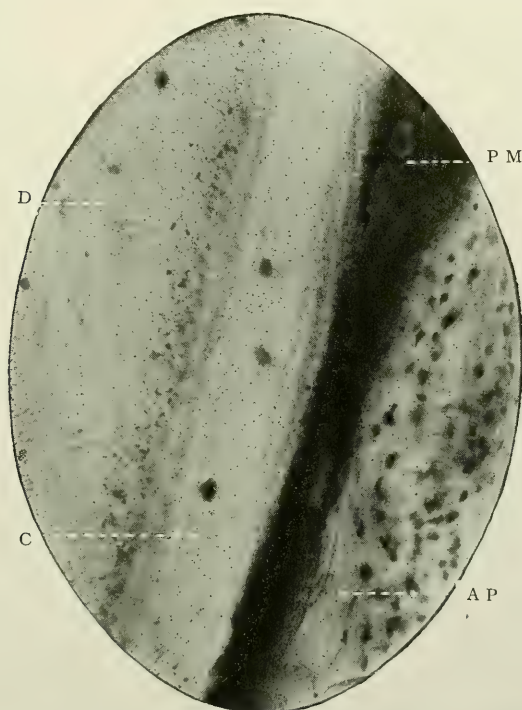

FIG. 203.—Cementum, showing its hyaline character and absence of lacunæ. the three dark masses being foreign bodies on the surface of the tissue. D. Dentine; C. Cementum; P.M. Periodontal membrane; A.P. Alveolar process. Magnified 250 times.

scopical one.   These hypersensitive surfaces very frequently, indeed, develop into acutely esthetic cavities.   Whence comes the pain? and what is found on histological examination?   A tiny strip of dentine uncovered by the other hard tissues.   The anatomical relation-ships of the hard tissues at the necks of teeth are as follow: (*a*) Enamel overlaps the cementum in about .5 per cent. of cases.   (*b*) Cementum overlaps the enamel in 60 to 65 per cent. of cases. (*c*) The

two tissues meet *bout à bout* in 30 per cent. of cases. (*d*) Finally, they fail to meet at all, and leave the peripheral surface of the dentine exposed, in 5 to 10 per cent. of cases.

Thus normally, it would appear, that the two tissues are in absolute contact, and both lie in the same plane without any involution whatever (see Chap. III, Vol. 1).

Now, enamel being outside the pale of nutrition, and once formed always formed, and cementum itself being insensitive, it is clear that the dentinal tubules are actually exposed at their distal extremities, and their contents are irritated by the pressure or presence or friction of a foreign body in that region. Enamel is nerveless, and cementum in normal conditions does not contain any elements of a nervous or protoplasmic character in sufficient amount to induce pain in suitable circumstances. No doubt prolonged irritation might lead to exalted sensibility on the part of the pulp, and if decalcification of the thin edges of the enamel and cementum takes place, with loss of substance, an erosion cavity is produced.

(2) *Thermal sensations* are easily explained, owing to their being due to a raised or lowered temperature of the pulp through conduction on the part of the enamel—a state of things which the pulp immediately recognizes, by means of the dentinal fibrils. It does not always happen that a pulp is painful when icy-cold or hot water is placed on the enamel. When it is, however, it is either hyperæsthetic, or perhaps more correctly, hyperalgesic, and is more readily affected by outside influences than it should be, or the sensorium is more appreciative than natural of the impressions conveyed to it from the pulp.

Occasion arises sometimes when a devitalised tooth whose pulp has been removed gives the impression to the patient by pain that it is still present and alive. The exhaustion of air around the neck of the tooth—that is, an alteration in or lowering of the local temperature on the part of the patient—gives rise to pain. On examination it may be found that the interdental gum may have been inadvertently removed while operating on the adjacent tissues, and the free edge of the periodontal membrane nearly exposed. It is therefore probable that the root membrane transmits the sensations of pain to the brain, which interprets it as that coming from the pulp. With regard to those cases of a root or portion of a root which, having been denuded of its alveolar socket and mucous membrane, such as is frequently observed in connection with the palatal roots of maxillary molars, is very sensitive to thermal changes, and is

rendered insensitive by means of the application of silver nitrate, the author is not prepared to state what really occurs. Cementum is incapable *per se* of transmitting sensations; and "the living chain of protoplasm" which Bödecker so deftly described as passing from the dentinal fibrils through the granular layer of Tomes to the lacunæ and canaliculi of the cementum, and so on to the nervous bundles of the periosteum, is a myth. For a solution of silver nitrate cannot
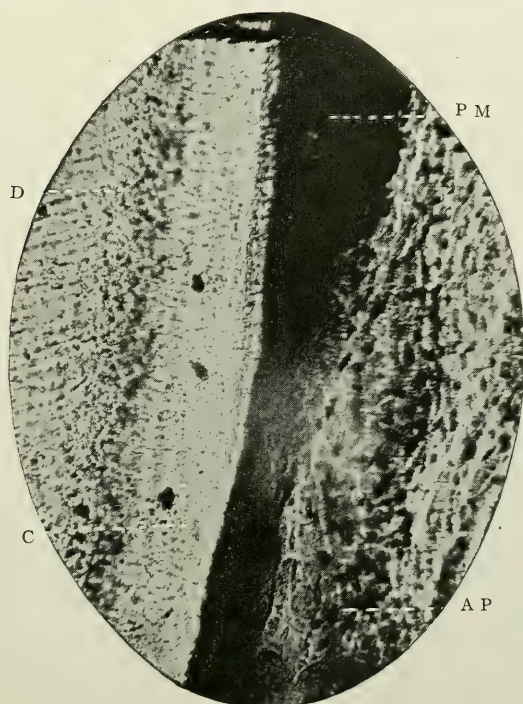

Fig. 204.—Same as preceding, but differently illuminated, to show granular character of cementum.

soak through cementum whose free layers are almost homogeneous in character and hyaline in structure (Figs. 203 and 204).

If a pulp is frequently painful when hot substances and fluids are taken into the mouth, it indicates that, though there may be no apparent lesion, it is certainly on the "down grade."

(3) *Chemical Stimuli.*—The application of lukewarm mineral acids or alkalis to an unbroken surface of the teeth gives rise to no pain in the pulp.

(4) *Electrical impulses*, however, are full of interest. "Electric

cells are frequently formed in the mouth, metallic poles being present and an electrolyte intervening. The greater the electro-positive or electro-negative the metal, the greater the electro-motive force. Now, in the electrolytic scale, gold is nearly the most electro-negative of all metals placed in the mouth; while aluminium is the most electro-positive. If, therefore, two metals of different electromotive force come into contact or almost into contact, and the saliva is ionized to such an extent as to be efficiently electrolytic, the cathions, or — ions, will move toward the + pole or more electropositive side, while the anions, or + ions, will move toward the — pole, and a certain amount of electricity will be evolved.

"This action may be carried to a greater degree than is usual in the mouth. Clinically this force becomes more manifested, at times, under certain conditions, in a disastrous manner. Thus a 16-carat gold band which is used for supporting a denture, if attached to a molar tooth, for instance, which contains a large amalgam filling, may in these cases become so electrically affected as to break, on account of the molecular changes set up by the current, and the filling similarly to become disintegrated and fall out.

"Mere contact of a metal with enamel or dentine is not appreciated by the pulp. It is when two dissimilar metals are brought into contact, separated only by a thin film of saliva, which acts as the electrolyte, and the circuit is completed, that voltaic currents are set up—as for instance when the metal ring of a small mirror touches the surface of a gold or an amalgam filling. This, of course, constantly takes place during operations, and is as a rule unnoticed by the patient, on account of its extremely light character; but at times the pulp, stimulated by the electrical action, responds by a sudden spasm of acute odontalgic pain." ("An Introduction to Dental Anatomy and Physiology" 1913.) Is the pulp normal in these latter fairly rare cases? Probably not—it is beginning to undergo morbid changes; for the phenomenon may be observed, as far as clinical experience goes, equally beneath large as well as less bulky fillings. The pulp is not hyperæmic; it is hypersensitive, or particularly "receptive,"—owing, no doubt, to an exalted condition of the cerebro-spinal nervous system which happens to be synchronous with the electrical phenomena. Tests as to the acidity or otherwise of the saliva are unattended by success. Here, of course, mere contact with the filling alone induces no response; the circuit must be completed, either by simultaneously touching filling and tongue or filling and buccal mucous membrane.

It is interesting to note that the same degree or kind of reaction of the saliva may not obtain simultaneously in the same mouth. Thus some portions of the gums or buccal mucous membrane may show, at the same moment, an acid, an alkaline, and a neutral reaction to litmus paper.

(B) REFERRED PAIN AND OBSCURE REFLEX ACTS

Entirely different from these local manifestations are those nerve pains set up in apparently sound teeth on holding lukewarm saccharine substances in the mouth.

A particularly sweet body, for instance, will at times, when the functions of the central nervous mechanism are exalted, give rise to intensely acute pain which may last an appreciable length of time and then passes slowly away. This is no mere gustatory hallucination, no co-ordinated association of ideas, but a physiological fact. Here is a reflex act. It is impossible for sugary solutions *per se* to so rapidly attack enamel or cementum or even exposed dentine as to induce these pains. The length of time required for the paroxysm to make itself felt after the application of the stimulus is about 0.05 to 0.06 of a second. It is not through the hard tissues that the impulse passes, it is through the nerve supply of the pulp. The afferent nerves are those sensory branches of the Fifth which supply the oral mucous membrane, and the gustatory and glosso-pharyngeal, with possibly the *Chorda tympani;* and it is the act of placing the saccharine-bearing material on the tongue and inside of the cheek that brings about the reflex act. This, too, only occurs at times, when probably the sensorium is especially liable to receive and transmit slight impulses to teeth, the pulps of which, while not degenerated are certainly not entirely normal.

There are many examples of the pulps of teeth causing pain when there is no obvious lesion. Occasionally the eating of very sweet or sour bodies will set up excruciating synalgia in sound teeth. Through the glosso-pharyngeal and the *Chorda tympani* nerve sensations are felt by the brain, which as in the case of the amputated foot makes an error of judgment as to the position of the stimulus. In its essence it is akin to mysophobia and certain other systemic morbid emotivities.

Reverting for a moment to this question as an aid to the proper understanding of this reflex act, we know that mysophobia, or the fear of improper or inapposite contacts is not confined to manifes-

tations in the form of a natural aversion to touching disagreeable bodies, or those capable of undergoing decomposition. Many people cannot bear to perceive, by the sense of touch, the surface of fabrics such as velvets or silks, or the external parts of the skins of fruits such as peaches. There may arise in the minds of some, fear of contact with metallic objects, such as copper, pins, and needles, or, according to Morselli, pieces of glass. It is possible that derangements of the tactile digital sensibility are partly the cause; for Marcé has described a case in which, through a fear of seeing pins or needles adhering to the fingers, complete insensibility of the skin of the hand was produced. Among other illustrations of systemic morbid emotivity, Morel, quoted by Dr. Charles Féré in his "La Pathologie des Emotions," 1899, cites the following: King James II of England trembled at the sight of a naked sword; Erasmus suffered from febrile fits on seeing a plate of lentils; Scaliger was seized with nervous tremors at the sight of a carafe of water; Bacon experienced the state of syncope during every lunar eclipse.

"SETTING THE TEETH ON EDGE"

The complex series of phenomena constituting that anomaly of sensibility popularly spoken of as "setting the teeth on edge" may perhaps be placed in the same category as the preceding illustrations, for, in this case, it is partly the outcome of an innate antipathy on the part of the individual. It is something more than a mere reflex act, inasmuch as it is accompanied and complicated by a mentality which is the effect of the workings of a higher conscious cerebration. This syncinesia, among others studied by John Hunter, Müller, and Gubler, which is purely physiological, or perhaps borders on the abnormal, may be caused partly by the action of the auditory nerves, and partly by the gumaisthenic or somaisthenic perceptions of the person.

Gumaisthenic Perceptions.—The phenomenon was known to the Prophets of old whose words, recorded in the Bible, are as follow:

Prov. x, 26: As vinegar to the teeth, and as smoke to the eyes, so is the sluggard to them that send him.

Ezek. xviii, 2: What mean ye, that ye use this proverb concerning the land of Israel, saying "The fathers have eaten sour grapes, and the children's teeth are set on edge?"

Jer. xxxi, 29, 30: In those days they shall say no more, The fathers have eaten a sour grape, and the children's teeth are set on edge. But every one shall die for his own iniquity: every man that eateth the sour grape, his teeth shall be set on edge.

Ousaisthenic Ideas.—Shakespeare declared:

> I had rather hear a brazen canstick turned,
> Or a dry wheel grate on the axle-tree;
> And that would set my teeth nothing on edge,
> Nothing so much as mincing poetry.
>   —*1 Henry IV*, iii, I.

Through the action of the auditory nerves the grating of a dry wheel or stick of schoolboy's pencil on his slate may induce it; and not infrequently the tactile sense. When the fingers are rubbed lightly on the surface of a piece of velvet, this curious sensation of pain in the teeth is noticed. *Somaisthenic* ideas become converted into a modified form of acute odontalgia. If the fingers are rubbed on a piece of glass-paper or a hot metallic surface, the mind appreciates the condition thus induced locally, and pain is felt, not in the teeth, but in the digital extremities themselves.

The explanation of the conversion of the somaisthenic perceptions into pain would seem to be the following:

The simple act of touching the velvet does not *per se* induce pain, and would not do so if the mental attitude of the individual were not an anticipatory one. He has the knowledge that his teeth will ache, and that possibly a cold tremor will pass down his spine, as his fingers are brought into contact with the velvety field. It may be that the brain is unaccustomed to determine the amount of muscular effort set up by the digital movements, or that it is disturbed by the unusual and somewhat naturally repellant act. There is no doubt that there is a great limitation to the capacity of the synthetic action of our minds in appreciating the changes which occur within our bodies, and the mechanism of the brain frequently fails in its estimation and correct interpretation of what is going on.

Other kinds of obscure reflexes, not due to pathological causes, may be cited. Pressure on the supraorbital branch of the frontal division of the ophthalmic nerve may induce pain in a maxillary canine. An observer tells the author that when, in India, he has witnessed children sucking sticks of sugar-cane, he himself has had toothache.

So it is obvious that the optic, the auditory, and more than all, the trigeminal nerves, are closely associated with these reflex acts in health as well as in disease, and that as diseases of the teeth, caries and the like, will produce reflex disturbances in the organs and parts supplied by them, so they, conversely, under certain forms of irritability will set up odontalgia.

15

RECEPTIVITY OF THE PULP: ITS HYPERÆSTHESIA AND
DYSÆSTHESIA

But ordinarily no pain is felt; when it is, however, the condition of
the pulp is that of dysæsthesia, and comes on only when the central
and peripheral nervous systems are in a state of excitability or
receptivity.

The physician is often brought face to face with other varieties of
hyperæsthesia, such as gastralgia, enteralgia, the epileptic aura, and
so on. And the term dysæsthesia is used in connection with the organs
of special sense, and the condition indicated by the appearance of
subjective phenomena referable to these organs—"of the eye,
by the appearance of sparks and flames; of the ear, by the perception
of sounds, such as humming or buzzing; of the nose, by the percep-
tion of odors; of the tongue, by the perception of flavors" (Bris-
towe), and finally, of the teeth, by the recognition of those interest-
ing reflexes to which allusion has been made.

(c) DISTURBANCES OF THE VASCULAR SYSTEM IN THE PULP

Allied to these morbid states, but totally different in origin,
are those other conditions a few particulars of which may be now
mentioned.

Odontalgia of a severe type may be induced by morbid conditions
of the blood, which circulating through the vessels of the pulp
produces pain and organic change in that tissue.

Increased intradental blood pressure. To Dr. Ferdinand Tänzer
(*Oesterr-ung. Vierteljahrsschrift f. Zahnheilkunde*, 1907) belongs the
credit of first drawing attention to the effects of increased blood
pressure in the pulp. If a pulp is very small, as the result of ana-
tomical overgrowth or developmental defects of the hard parts,
or is habitually anæmic as a result of systemic disturbances, any
rise of blood pressure in it may induce pain. It is first dysæsthetic,
then hyperæsthetic, and then hyperæmic, and lesions of the vascular
system may follow, leading ultimately, if long continued, to fibroid
degeneration, death, and gangrene.

The causes of these obscure forms of odontalgia may be divided
into (i) congenital and (ii) acquired.

The former are associated with puberty and the catamenia.
The signs and symptoms are as follow: Severe pain in apparently
normal teeth, generally occurring in girls and women, which are

agonizing in character and practically resistant to dental thera-
peutics. There is no obvious lesion.

If a tooth is removed and examined microscopically it may happen
that a marked congenital defect can be noticed. If the pulp is too
small for the tooth—if the area of the pulp is diminutive and that
of the hard parts unduly large, indicating that enamel and dentine
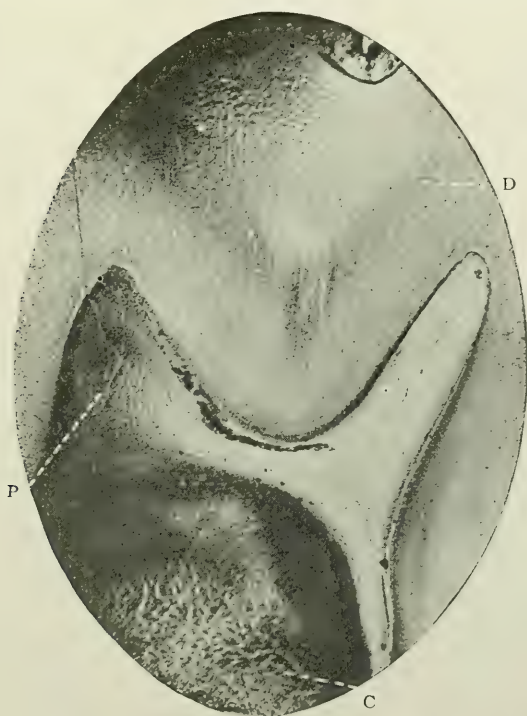have been produced at the expense of the soft tissues (as in Fig.

FIG. 205.—Longitudinal section of molar showing small size of pulp and great
thickness of hard parts. (History of tooth narrated in text.) D. Dentine; P.
Pulp; C. Cementum. Magnified 12 times.

205)—the increased blood supply would probably be attended by
pain, partly because the small size of the pulp reduces its trophic in-
fluences on the tooth itself, partly because of the hydrostatic conges-
tion that has taken place, partly because of the absence of a collat-
eral circulation, and partly because the character of the chemical
constituents of the blood is altered at these periods.

Thus, it has been proposed by Dr. Blair Bell, *Proceedings Royal*

Society of Medicine, 1908—who claims that the metabolic processes concerning the calcium economy exercise an all-important influence upon the genital functions and are necessary factors therein—that the well-known vaso-dilatation which occurs in the combs and wattles of laying hens is due to the drop in (*i.e.* loss of) the calcium contents of the blood, whereby a sort of chilblain condition is produced.

If vaso-dilatation or local hyperæmia can occur, as suggested, in the peripheral organs of the hen, it is conceivable that a rise of blood pressure in the pulp can also be produced in similar circumstances. Such a case, to which allusion has been made, occurred in the practice of a friend of the author, to whom he is indebted for the following particulars:

Notes on a Case of Obscure Odontalgia

"October 14, 1906. Miss A., aged fourteen. No caries. Pain experienced along the upper and lower jaws on the left side. All teeth were painful on pressure and slightly loose. Pain in joints. Saliva, very acid. Pain more intense during cold or damp weather.

"*Diagnosis:* Rheumatism of jaws.

"The patient was sent to a physician who reported: 'Patient suffering on October 24, 1906, from slight rheumatism and hyperchlorhydria. The latter was rapidly cured by the administration of magnesium peroxide . . . . . Later on (June 1907), when I saw the sections of the patient's teeth, I thought that the trouble might be due to deficiency of calcium salts and a lower coagulability of the blood. I therefore put her on a course of calcium lactate. I saw her three weeks later, when I ordered her to continue the treatment for a full six weeks in all, and have not seen her since.'

"The dental pain gradually departed.

"May 1, 1907: For four days patient complained of intense pain in the left mandibular second molar. Tooth tender on percussion; affected by temperature. Local and internal remedies of no avail for any length of time.

"*Diagnosis:* Presence in pulp of pulp nodules.

"May 7th: Extracted tooth: Too tender to bur out. Tooth submitted to microscopical examination; no pulp nodules on sectionising (see Fig. 205).

"June 7th: Great pain in left mandibular first molar; symptoms as before. It was thought the condition might be due to extra blood

pressure in the pulp.   Tried all local and internal remedies as before. The only thing that relieves the pain is abstraction of blood by leeches, but the effect is only for a few hours.

"June 20th: Extracted tooth.   Sent the patient again to the physician to have blood tested.

"Between last date and March 1908 patient complained of pain, at times in the left maxillary second molar, but the physician's treatment *after three doses* generally relieved the pain.

FIG. 206.—Longitudinal section of premolar of child.   P. Pulp; D. Dentine; P.N. Pulp nodule.   Magnified 12 times.

"March 12, 1908: Great pain experienced in left maxillary second molar; insomnia.   Applied leeches, with some relief.   Found a small pinhole cavity, but drilling gave rise to intense pain.   Patient had an anæsthetic and then the pulp was drilled into.   Great relief followed, accompanied by much hæmorrhage.   This having ceased, I removed the pulp under cocaine pressure anæsthesia.   The root-canals were very small.   Filled them with light dressing, with temporary gutta-percha in cavity.

"March 19th: Filled root-canals and filled the tooth.

"June 11th: Great pain all night in the tooth. Applied aconite and iodine, with no result. Drilled into canals left open, then applied a light dressing.

"June 12th: Pain getting worse; patient up all night; tooth very tender. Extracted tooth. Canals were all open; two roots covered slightly with glairy exudation, one of them having a slight nodule and slight absorption.

Fig. 207.—Longitudinal section showing fibroid degeneration of pulp in deciduous incisor. D. Dentine; P. Pulp; A. Abraded area of dentine. Magnified 12 times.

"The gums were always normal—never injected or tender, except in the last instance, when they were slightly tender."

PHASES OF DEGENERATION

Acquired lesions are those of the vessel walls induced by such systemic disturbances as are found in anæmia, chlorosis, gout, maras-

mus, etc.   Here more or less permanent injury is done to the coats of the arteries and veins, resulting in hæmorrhage, thrombosis, chronic endarteritis, calcareous degeneration, and so on.

If calcareous degeneration in the form of pulp nodules (see Fig. 206) or attached new growth occurs, and is profound, the symptoms are similar to those of developmental origin, but the result of the treatment is not the same, inasmuch as extraction of the apparently sound tooth immediately cures the odontalgia.

If the former conditions exist there is little if any pain complained of.   The patients are young children, but the pulps are approaching senility, as is evidenced by their exhibiting various phases of reticular atrophy or fibroid degeneration (see Fig. 207).

This degeneration is extremely common, and is probably due, as a complication, to thrombosis of the capillaries and veins, and as a result the impairment of the vaso-motor mechanism, which leads to vaso-dilatation and diseases of the vessel walls generally.

OBSCURE CAUSES OF ODONTALGIA

Odontalgia may thus occur in teeth unaffected by dental caries. It is then due to one of the following causes:—A. Increased or diminished blood pressure in the pulp; B. Pulp nodules; C. Altered chemical constituents of the blood; D. Intra-oral electrical impulses; E. Reflex from the tongue; F. Lesions of Vth nerve; G. General neurasthenia and debility.

In conclusion, these remarks on non-carious and non-apparent lesions of the pulp may be epitomized by saying that, as this organ is influenced pathologically by general diseases of the nervous and vascular systems, obscure cases of odontalgia should always be regarded from the standpoint of the physician as well as of the dental surgeon; and that, if there is a marked personal or family history of gout, rheumatism, or allied conditions, pulp nodules may be diagnosed; if the patient is anæmic, chlorotic, or marantic, or recovering from a long febrile disease, early stages of fibrosis may be suspected; also that hyperæsthesia or dysæsthesia is indicated when a patient is neurotic, or neurasthenic, or subject to "nerve storms," or is suffering from a form of nervous excitability or exaltation, or perhaps exhaustion.

CHAPTER IX

THE VASCULAR LESIONS OF THE DENTAL PULP

Introductory—Anatomical considerations—General effects—Histo-pathology—Causes—Clinical significance.

INTRODUCTORY

Of all the different tissues that go to form the component parts of a tooth, the pulp is the most interesting. It is likewise the most important, for on its vascular and nervous mechanism depends the vitality and therefore the utility of each unit of the masticatory apparatus. The strikingly singular character of the anatomical distribution of the vascular supply arrests attention, and the impartial observer, who might be suddenly called upon to study fully and critically the diseases of the pulp as an entirely new field for his own exploitation, would probably note that fact at the commencement of his laboratory experiments and experience.

ANATOMICAL CONSIDERATIONS

Two facts stand out pre-eminently as being of great significance— (1) the absence of a collateral circulation in the pulp itself, and (2) the non-valvular character of the veins, which at the same time are non-collapsible.

(1) With regard to the former, it may be recalled that during the development of the teeth the tissues formed from each layer of the primitive blastoderm are supplied by two separate groups of vessels, of which the internal set, distributed to the mesodermic structures, vascularize the dentine papilla, the dental sac, and the surrounding bone. In adolescent and adult pulps the branches of the internal set enter the apical foramina of the teeth usually as a single trunk, which may measure as much as 83μ in width, to become almost immediately bifurcated and divided into many subsidiary branches, ending near the basal layer of Weil in an anastomosis of capillary loops whose individual lumina are roughly, on the average, about 8μ in diameter (see Chap. VII, Vol. I).

If the lumen of the main artery, entering the apical foramen, is by any means occluded, the integrity of the whole of the vascular system is imperilled, as there are no collateral branches to restore the balance in the blood-stream.   This actual condition is almost if not quite unique in the general anatomy of the body.

These remarks apply with no element of uncertainty to the vascular system of the single-rooted teeth.   It cannot, however, be asserted with equal confidence that the pulps of molars have no collateral circulation.   The exact method of distribution in these larger teeth has never been ascertained, owing to the difficulties encompassing the performance of an artificial injection of the vessels in adult age.   But clinically it would seem to be impossible for a collateral anastomosis to exist; and from an embryological point of view the statement of Lepkowski may most probably be relied upon.   This observer shows (*Anatomischer Hefte*, 1901) that "In the germ of the two-cusped tooth there are present two bundles of vessels separated from one another," etc. (see Chap. XII, Vol. I).

(2) Again, on account of the patency and non-valvular character of the veins it may be assumed that exogenetic influences in normal circumstances cannot affect the flow of blood in the pulp.   The veins are not subjected to muscular or other external pressure, and in this respect agree with other similarly constructed tubes, such as the superior and inferior vena cava, the pulmonary veins, and those in the interior of the cranium and vertebral column, and long and short bones.

Hence it is observed that these two facts are conspicuously apparent, the first being probably of the greater importance, as the general systemic veins that are 2 mm. and less in diameter are un-provided with valves.   But the significance of the absence of a collateral circulation on the causation of diseases of the pulp cannot be overestimated by the perspicacious and discriminating student.

GENERAL EFFECTS

After due consideration of these anatomical conditions it is not a matter for surprise that lesions or degenerations arising from varia-tions in the blood current or in the blood pressure in the dental pulp should be fairly common.   So much is this the case that one would probably be not far from the truth in asserting that, while teeth are so very frequently the victims of dental caries, they may also equally and simultaneously be subjected to internal retrogressive

FIG. 208.—Longitudinal coronal section of premolar with pulp *in situ*, showing thrombosis of capillaries and other vascular lesions. Prepared by the Author's process; stained with Ehrlich's acid hæmatoxylene. A. Branch of main artery; V. Enlarged vein; C. Capillary filled with thrombus; T. Thrombus becoming organised; F. Early stage of fibrification of pulp tissue proper; O. Odontoblast layer increased in width and in the number of its cells. Magnified 45 times.

changes induced by a lowered or altered physiological resistance on the part of the pulp, through the unusual characteristics of its blood supply.   This does not apply entirely to the normal individual, but more especially to those persons who suffer from disturbances of the circulatory system.

It is well known that many persons undergo a certain temporary discomfort brought about by hyperæmia of the pulp.   For some reason or other the vessels become vicariously over-filled and undergo hydrostatic congestion, which presently disappears when the cause is removed or when there is a good outlet provided for a free flow. If, however, the intradental pressure is so severe as to prevent the occurrence of an efficient and quick relief, then the tissues degenerate and perhaps die, as they are unable, on account of their dentinal environment, to accommodate themselves to their engorged state.

Tänzer (see Chapter VIII) records a case—among others—where, on account of the circulatory irregularities in the internal genital organs and adnexa of a patient, the pulp of a tooth died as the result of the abnormally increased intradental blood pressure. The introduction of metal fillings and cement floorings into carious cavities is sometimes followed ultimately by local pain, and the work of obturation is credited by the uninformed with being the cause of the odontalgia, while it is merely often due to either reflex nervous irritation or a rise in the local blood pressure.   The writer just quoted proceeds to summarise his remarks by saying that this increased intradental blood pressure may arise as a consequence of diseases due to circulatory deflections from the normal, to conditions of high nervous tension as in hysteria, to influenza, pregnancy, or occasionally as the result of traumatism.

It is thus clear that temporary engorgement of the vessels in the pulp tends to produce odontalgia of varying degrees of severity; but if this congestion be continued it leads eventually to death of the parts and cessation of pain.   This may be brought about (a) slowly, when the tissues pass through the various stages of fibroid degeneration, or (b) rapidly, when moist gangrene supervenes as a result of thrombosis, and arterio-sclerosis and sudden death *en masse* takes place.

It is believed that slight rise of blood pressure produces no symptoms of neuralgia, though it can be readily conceded that nerve pain from other areas may be reflected to a tooth which is sound but whose pulp is somewhat hyperæmic.

At the time that Dr. Henry Head wrote his remarkable observa-

tions on "Disturbances of Sensation, with Especial Reference to the Pain of Visceral Disease," *Brain*, Pt. III, 1894, and drew attention to certain well-defined areas of superficial tenderness of the skin, the study of the vascular lesions of the pulp had not been undertaken. His investigations were solely concerned with carious teeth, and showed that "As long as the pulp-cavity is not exposed the pain remains local. The patient will complain of an aching tooth and will point to the one affected." Local stimulation produces local pain (odontalgia), and "is exactly analogous to that produced by injuries to the conjunctiva or outer layers of the cornea." When, however, the pulp is exposed, the pain alters in character and distribution (neuralgia). "It starts in the affected tooth, and darts or shoots into the face, forehead, neck, or ear" (page 407). It is practically certain that, to-day, increased intradental blood pressure in sound teeth would be added to Dr. Head's previous classification.

Fortunately, gangrene as a permanent termination of thrombosis is a contingency of comparatively infrequent occurrence from a clinical point of view, and it is with certain atrophic changes, slowly produced, that the author desires to deal in the present chapter.

In Chapter VII the author gives an account of that common lesion of the pulp which is to-day spoken of as fibroid degeneration or reticular atrophy. Its etiology is, there, not thoroughly elucidated. The opinion is, however (see page 188), expressed that it represents "the natural and usual termination of the life-history of the dental pulp" occurring in aged teeth. The author has enlarged his views on the subject, for he believes that the senility of the pulp does not at all depend upon its age. Children may at times possess senile pulps, in the same way that the eyes of young persons often exhibit the *arcus senilis* of the cornea. Much of the material examined for the purpose of this Chapter was removed for the treatment of irregularities in position of the teeth, and many sections show that the whole of the dentinal wall was not completely calcified.

Probably this fibrosis or sclerosis is due, as a complication, in the first place to thrombosis of the capillaries and veins, and permanent dilatation through loss of tone (due to impairment of the vaso-motor mechanism) or disease of the walls of the arteries, with or without tiny hæmorrhages. This seems often to be succeeded or accompanied by a condensation or fibrification of the pulp tissue which lies between the basal layer of Weil and the substance of the
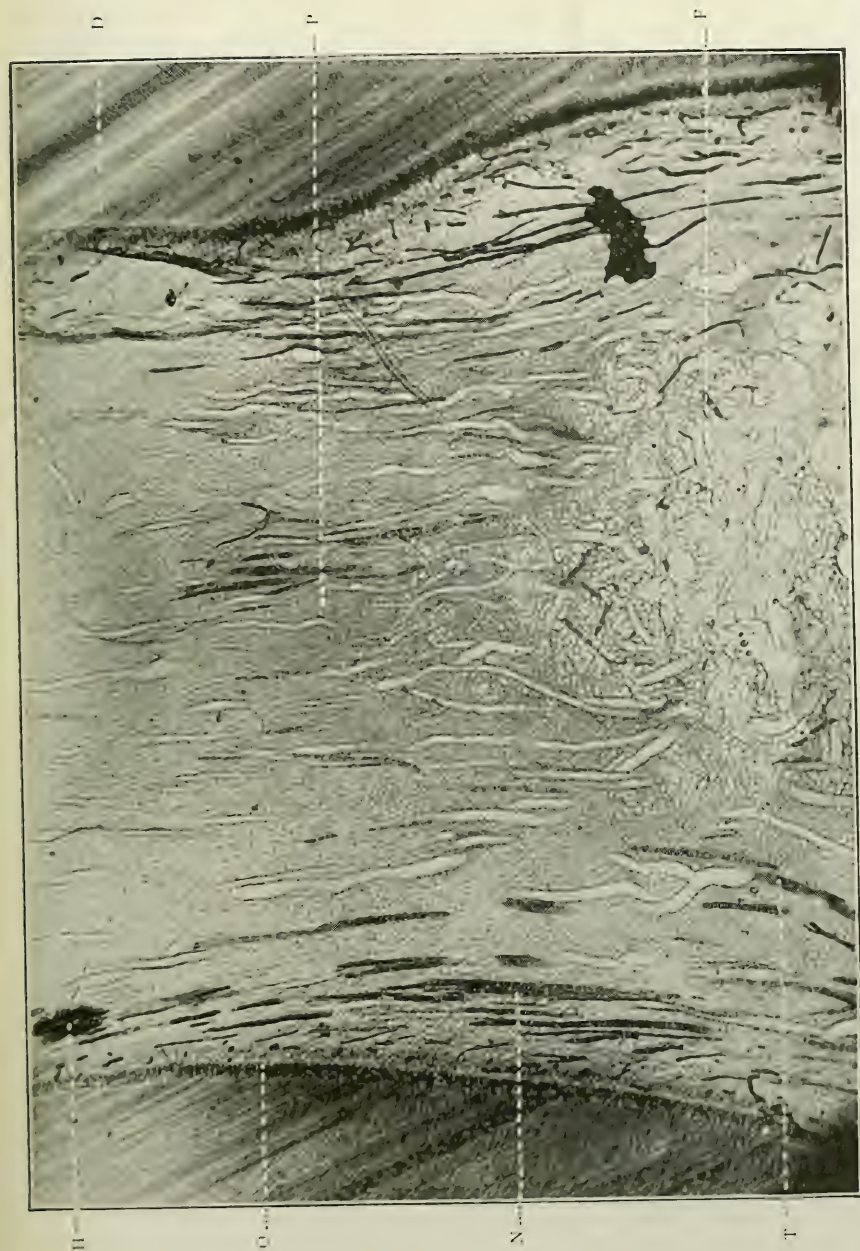
Fig. 209.—Similar to the preceding, through the cervical region of the pulp, showing transitional stages through which pulp passes. P, Pulp tissue passing from the fairly normal condition to a state of fibrillation and reticulation. F, Fibroid degeneration nearly completed; T, Thrombosed capillary; N, Degenerated bundle of nerves; H, Hæmorrhagic infarct; O, Odontoblasts; D, Dentine. Magnified 45 times.

pulp proper. A hyperplasia of the connective tissue fibres of the parts occur. "Sheaving" of the odontoblasts, with or without fatty degeneration, permanent distension of the arteries and arterioles, and rapid development of overgrowth of the fibroid tissue supervene, until a well-marked reticular atrophy appears, and in later stages complete fibrosis of the organ, with disappearance of all cells and nuclei, and every vestige of nerve bundle and vascular system.

HISTOLOGY

Longitudinal coronal sections of the pulps show to the best advantage these changes. The capillaries and small veins which are distributed to the peripheral parts are engorged. Very few are empty and none are stenosed. Under low powers of the microscope they present themselves as dark long strands running for the most part in the vertical axis of the tooth. The corpuscles and blood platelets which they contain are appreciably altered in shape and size—due, no doubt, partly to post-mortem changes, partly to the histological reagents employed—as these have not been specially directed to the preservation and staining of the blood elements— and partly also to those hæmic changes which favour coagulation, to be presently described. They may partially or completely fill the lumina of the vessels, and are sometimes arranged in rouleaux; but in addition have in places escaped from their walls as a consequence of rupture. Small arterial cauliflower-like hæmorrhages are seen frequently, at times among the odontoblasts, at times in the basal layer of Weil, and again at times in the substance of the pulp itself. The hæmorrhagic infarcts may vary in constitution from a punctiform collection of a dozen or more corpuscles, to a large mass, as in the photomicrographs. The endothelium of the *tunica intima* of these arteries and capillaries is altered, and the nuclei of its cells are indistinguishable. The larger arteries and larger veins are empty, as exemplified, and hyaline areas of degenerated material in many places extend across them, and as they become smaller occlude their lumina. The arteries have lost their distinguishing coats, and so have the veins, both classes of vessels having thinner walls than usual—a condition which pathologists would probably designate as hypoplasia or hyaline degeneration (Fig. 220).

The morphological effects of these vascular lesions on the surrounding soft parts is very noticeable. In some instances the

FIG. 210.—Cornual section through pulp of premolar showing probably one of the earliest phases of fibrosis. D. Dentine; Z. Zone of formed but not completely calcified dentine; F. Fibrification of pulp tissue, showing apparently a mutual relationship between length of odontoblasts and thickness of the band of fibrification; P. Large area of fibrification and condensation of tissue fibres and cells in body of pulp; V. Blood-vessels containing corpuscles but no thrombus; H. Small hæmorrhage among odontoblasts. Magnified 45 times.

odontoblasts, which are largely increased in numbers, are vacuolated and fatty (Fig. 221, o), the globules being often situated at their basal extremities, very numerous, and very small as a rule, but occasionally as wide as the cells themselves. They (the cells) are flattened laterally, and their nuclei are planiform. They are gathered together into bundles or sheaves (Fig. 212). The "sheaving" of the odontoblasts is of very common occurrence. It has been noticed by other writers, particularly by Walkhoff, who gives photomicrographs of
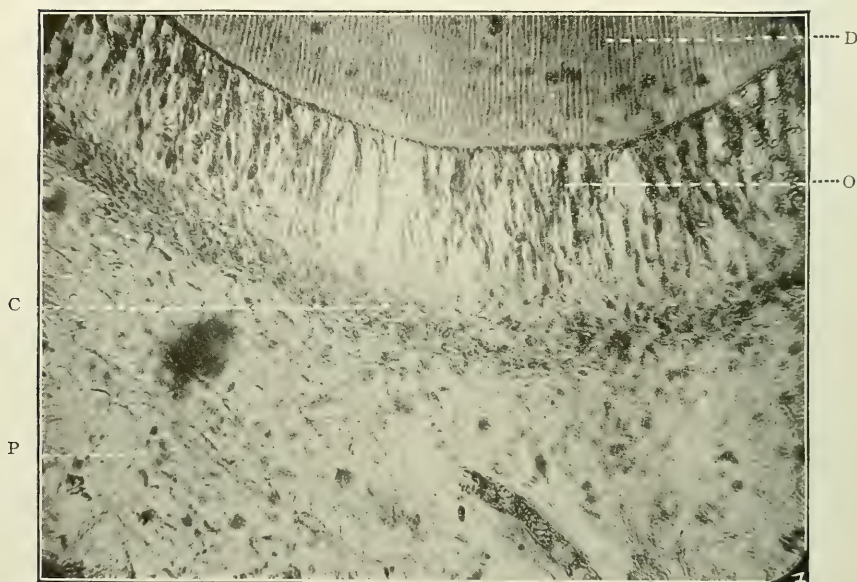
FIG. 211.—Details of early fibrification of pulp tissue. D. Dentine; o. Odontoblasts; c. Condensation of pulp tissue; P. Early fibrosis. Magnified 250 diameters.

it in his "Atlas of the Pathological Histology of the Human Teeth," 1903. The basal layer of Weil is curiously rich in small cells which have large round nuclei, and perhaps somewhat diminished in width, while its fibrous components are rendered more prominent and tough.

The cells of the pulp proper possess nuclei which are degenerate in shape and small in size; their branches are increased in number and extremely well marked. Here and there, in varying degrees of intensity, there seems to be a thickening of the intercellular cementing substances and chemical changes in the cytoplasm of

the cells.   When nerve bundles are visible, they appear to be degenerated also, and for the time being usually stain badly as long, thin, dark threads running alongside the vessels.

Organization of the thrombi is observed to be proceeding in places, leaving only a thin fibrous cord or hyaline plug coherent to the walls or completely filling it up.
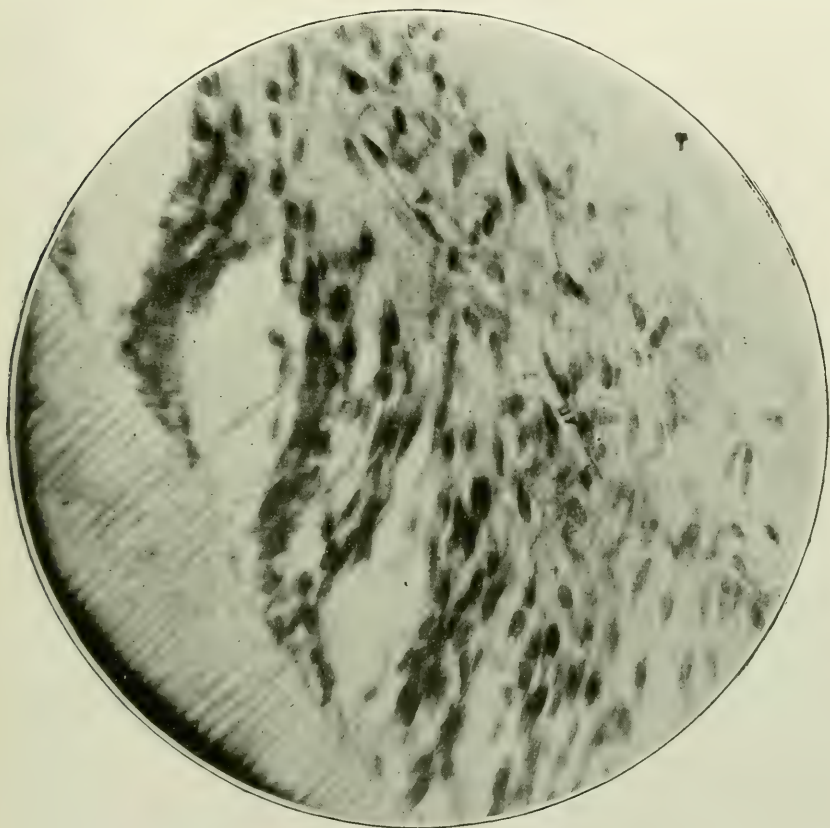
FIG. 212.—Degeneration of odontoblasts.   Magnified 500 diameters.

ETIOLOGY

Turning from the patho-histological aspect of this study, it is expedient and useful to inquire what possible factors have contributed to bring about this condition.   Why has the blood coagulated?   Why have the vessels become thrombosed and the soft tissues degenerated?   Probably the absence of a collateral circu-

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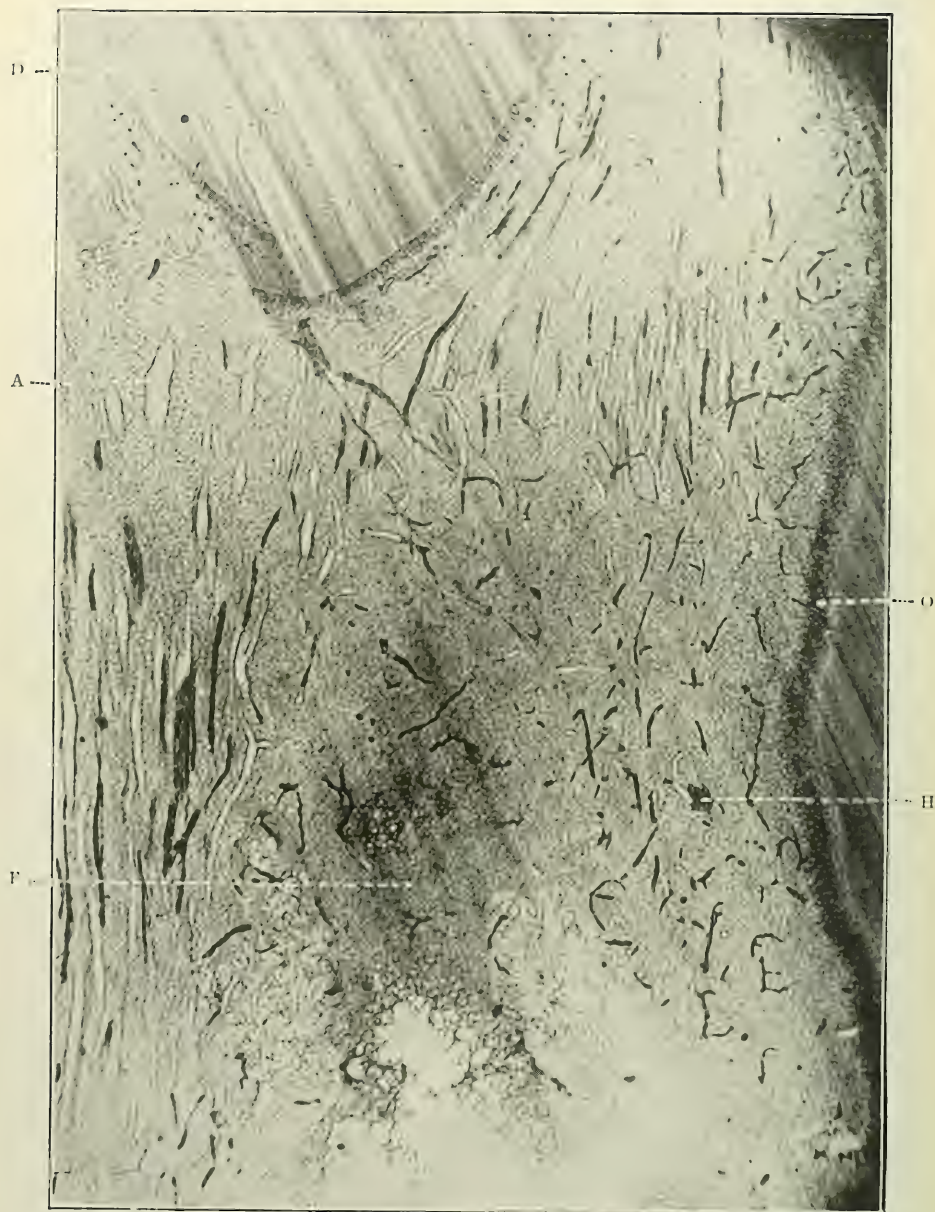
Fig. 213.—Similar to Fig. 208, showing a later stage of fibrosis of the pulp. D. Dentine; O. Odontoblasts increased in number; A. Arteriole; H. Hæmorrhagic infarct; F. Fibrosis of pulp.   Magnified 45 times.

lation predisposes to it; but its exciting causes cannot be so readily determined.   This absence of collateral circulation would predispose to the onset of thrombosis in cases, also, where any obstruction of outflow existed, *e.g.*, in cases of inflammation around the soft parts, or inflammation in the soft tissues themselves.   The vessels quickly become thrombosed, for instance, after the application
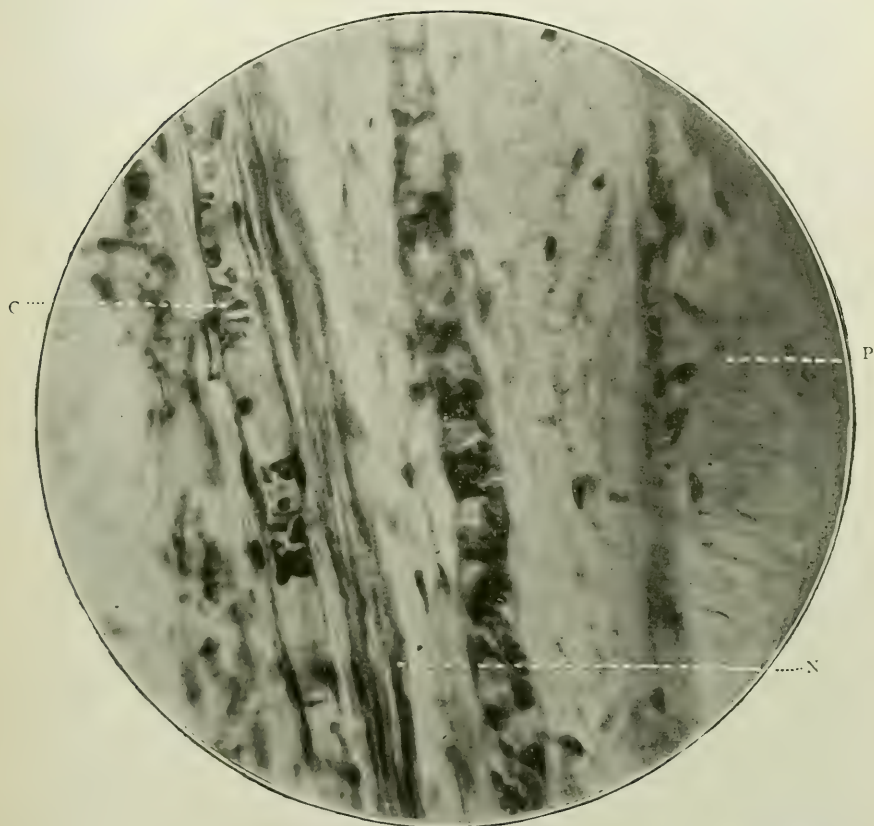
FIG. 214.—Details of blood-vessels.   c. Corpuscles adherent to vessel walls; n. Nerve bundles; p. Degenerated pulp tissue.   Magnified 500 diameters.

of arsenious acid to an exposed surface of the pulp, because the vessels are under hydrostatic conditions and inclosed in a resisting wall of dentine. Any increased volume of fluid (blood) must be compensated by a corresponding outpouring—as there cannot be an adequate displacement of the surrounding parts, owing to their circumscribed nature—to afford the room required. (It must not
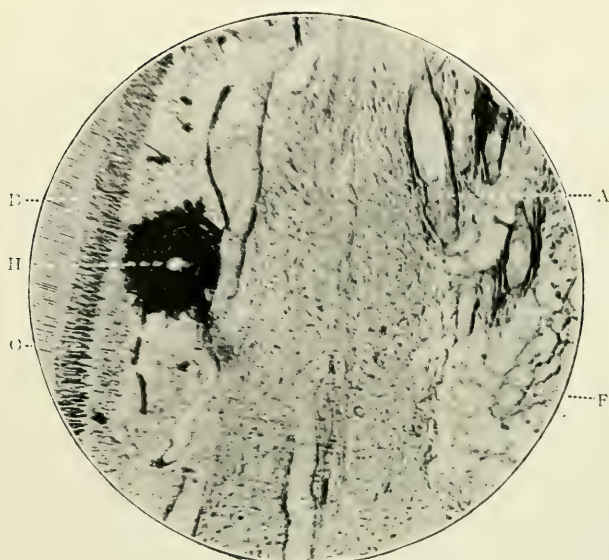
FIG. 215.—Pulp *in situ*. H. Hæmorrhage near basal layer of Weil; O. Odontoblasts; D. Dentine; A. Hyaline mass in small artery; F. Fibroid pulp. Magnified 250 diameters.
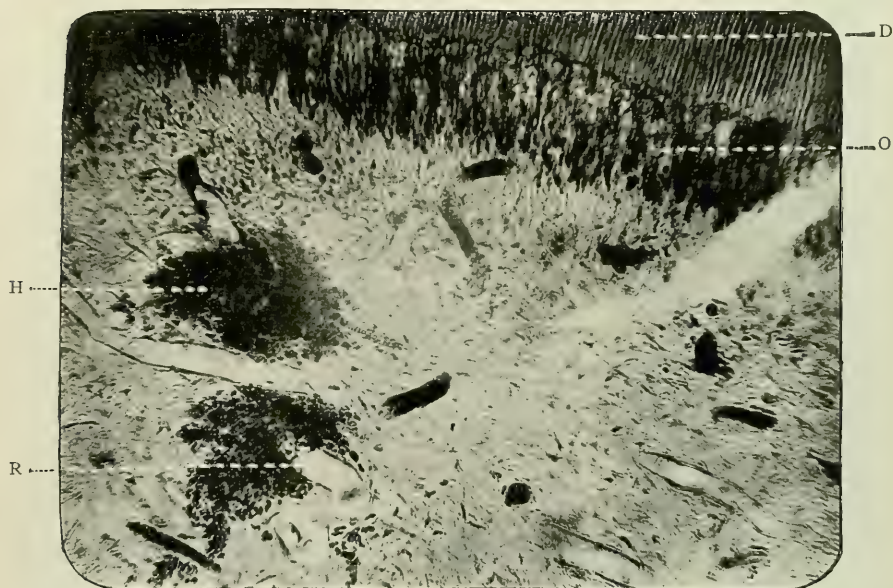
FIG. 216.—Similar to preceding (same magnification). H. Hæmorrhagic infarct; R. Ruptured artery whence blood corpuscles have escaped; D. Dentine; O. Odontoblasts.
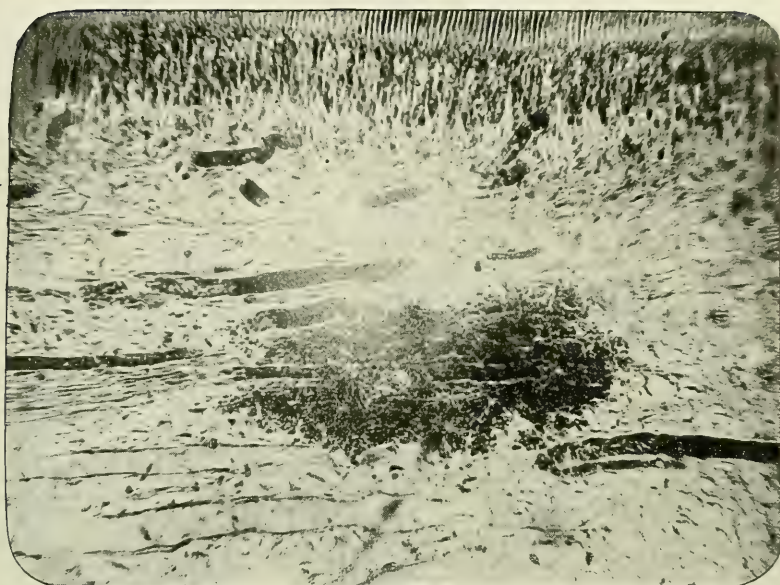
FIG. 217.—Similar to preceding (same magnification), showing hæmorrhagic infarct.
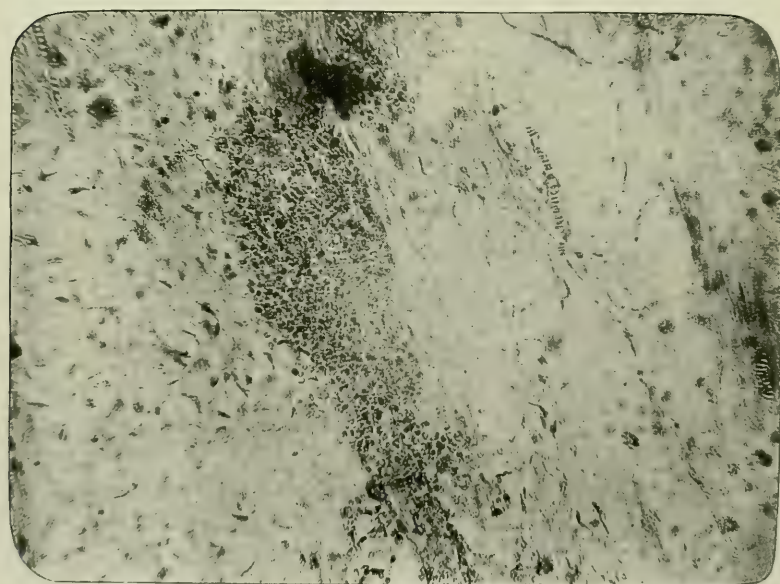
FIG. 218.—Similar to preceding (same magnification), showing hæmorrhagic infarct in centre of pulp tissue.

be forgotten, also, that no lymphatics have ever been identified as such in the pulp.) The thrombosis in this instance, which might almost be regarded as chemical or traumatic, is an early stage of acute inflammation, and is almost immediately followed by gangrene or death of the pulp *en masse.*

FIG. 219.—Hyaline degeneration of blood-vessels. Magnified 300 times.

A thrombus may be the cause or the result of arteritis or phlebitis. It may be due to chemical changes in the blood itself or lesions in the walls of the vessels, as in degenerations. Osler[1] has observed that in thrombus formation the blood platelets, fully investigated by Bizzozero, *Virchow's Archives,* vol. xc in 1882, and Eberth and

[1] Sir William Osler: Cartwright Lectures on the "Physiology of the Blood Corpuscles," 1886.

Schimmelbusch, *Virchow's Archives*, vol. ciii in 1888, are the first of all the blood elements to accumulate on the vessel walls during coagulation, and that the filaments of fibrin spread principally from these plate masses. They undergo viscous metamorphosis and also conglutination as explained by Thoma.[1]
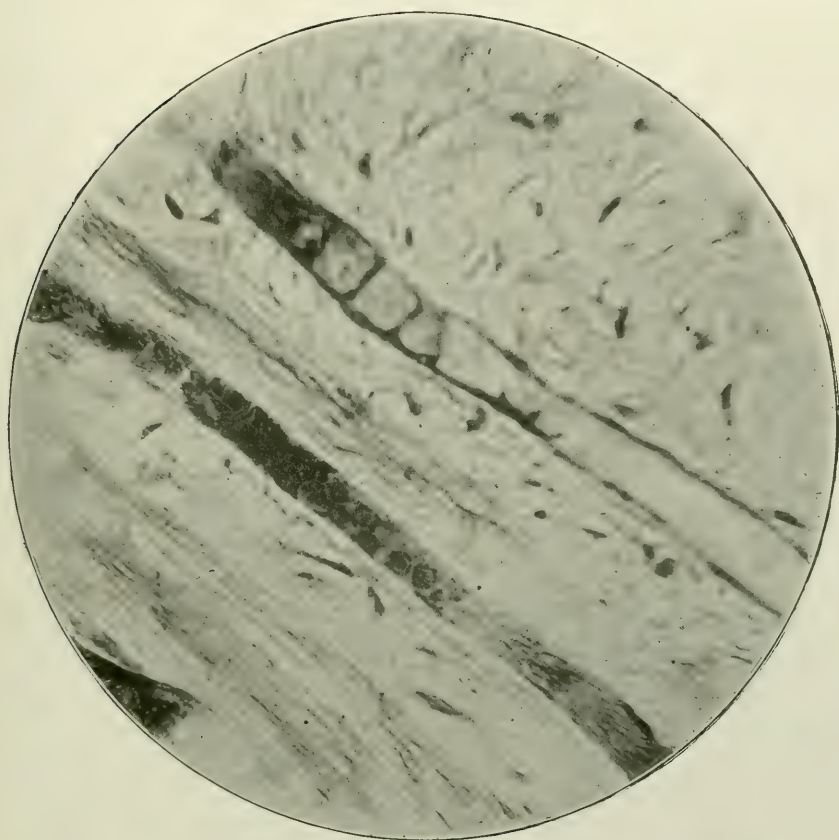
FIG. 220.—Thrombosis and hyaline degeneration of arteries.   Magnified 500 times.

The thrombi formed in the sections which form the subject of this chapter are obviously neither entirely the "red" nor the "white" varieties, but are clearly for the most part of a hyaline character, consisting of cells, platelets, fibrin filaments, and a colourless, semi-transparent, homogeneous material. They are entirely non-infected,

[1] Thoma: "Text-book of General Pathology," 1896.

although hyaline thrombi are generally associated with infected
conditions.

Thus it would seem that, in the dental pulp, chemical changes
in the blood, plus the unusual arrangement of the terminal vessels,
assisted by the *vis a tergo* which naturally leads to a certain amount
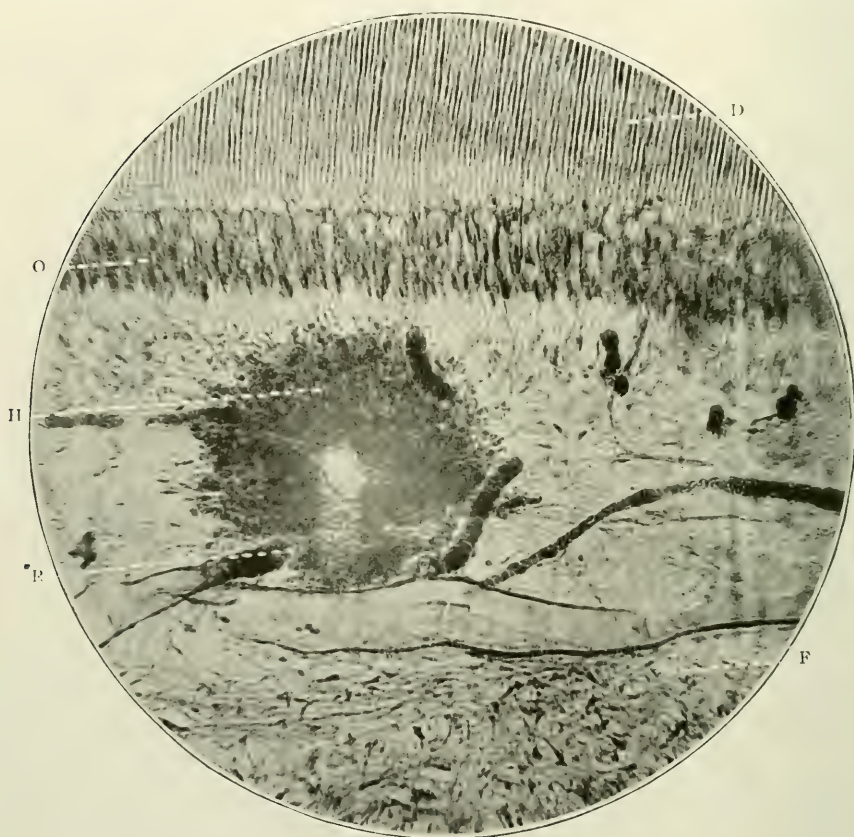
FIG. 221.—Details of Fig. 215 H. Hæmorrhagic infarct; R. Rupture of
blood-vessel; D. Dentine; O. Vacuolated odontoblasts; F. Early fibrosis of pulp.
Magnified 250 times.

of retardation of the flow and therefore coagulation—as first pointed
out by Virchow—are the originators of the atrophy, through loss
of trophic influences. The chemical changes are those undergone
by the blood through systemic derangements such as anæmia,
chlorosis, and those which take place toward the end of exhaustive
diseases.

The suggestion of infectivity can be at once dismissed, as all the sections which have come under the author's notice have been obtained from teeth, of the young and old alike, whose macroscopical aspects appeared to be sound.

It is of course well known that the blood of chlorotic patients may especially tend to produce diseases of the vessel walls—due, no doubt,

Fig. 222.—Reticular atrophy of pulp with thrombosed capillaries. H. Small hemorrhage near thin-walled vessel. Magnified 300 times.

to the great diminution of red corpuscles and the relatively greater number of leucocytes, and their slow movement along the walls of the vessels, together with an increased number of platelets. Marantic, anæmic, and debilitated conditions which often form the sequelæ of long-continued and enfeebling fevers and diseases,

probably also have the same or similar effects on the tissues of the
vascular system.

The cases here presented are entirely free from intrinsic calcifica-
tion, are unaccompanied by the formation of adventitious dentine or
any amyloid or fatty degeneration of the pulp; and in no specimen
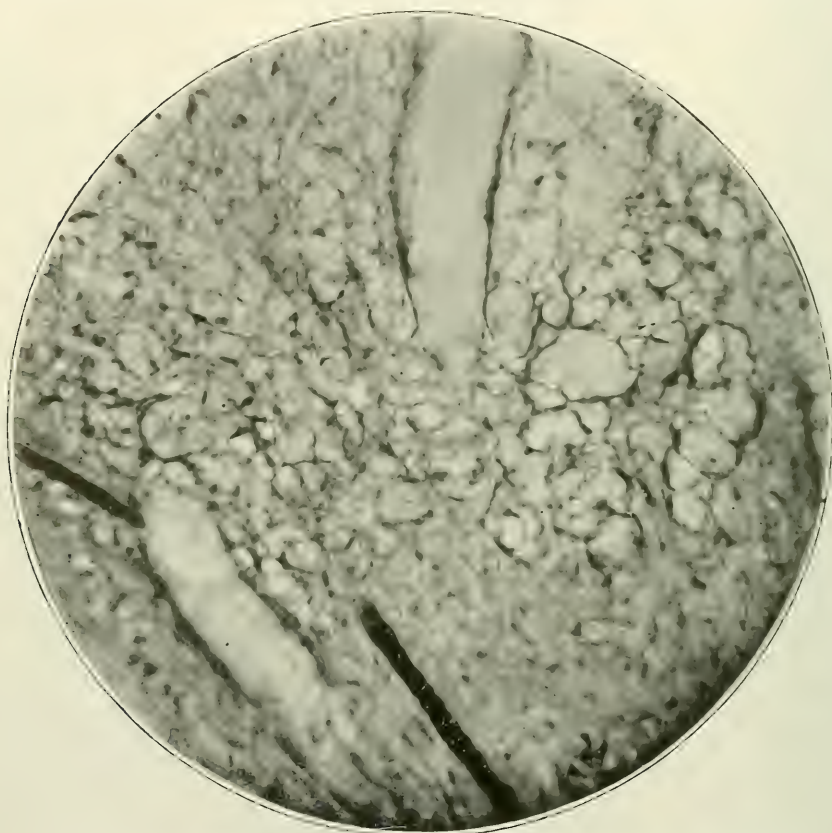
Fig. 223.—Nearly normal pulp passing into a reticular condition.   Magnified
300 times.

yet examined by the author have there been evidences of chronic
arteritis, atheroma or endarteritis, aneurysmal varix, phlebitis,
or varicosity of the veins.

CLINICAL SIGNIFICANCE

Of what value to the dental surgeon is a knowledge of the patho-
logical states sketched in the preceding pages?   Can it assist him

in the more perfect performance of his daily duties? Certainly; for it is at once obvious that if an attempt be made to "cap" an exposure of a pulp which happened to be diseased in this way, no matter how carefully or how aseptically the operation be done, no new secondary or adventitious dentine would be produced to

FIG. 224.—Details of thrombosis. c. Thrombosed capillary; a. Artery; n. Myelinic nerve bundle.   Magnified 250 times.

heal the breach of surface; and also the obstacles surrounding the complete devitalization of the pulp by means of arsenious acid, or its painless extirpation by means of pressure anæsthesia, become immediately manifest and indisputable. It is of course acknowledged that thrombosis and fibrosis are undiagnosable during life, but they may always be suspected in weak or marantic patients.

FIG. 225.—General view of pulp *in situ*, showing an intermediate phase of fibroid degeneration; longitudinal median section of canine. A. Thrombosed artery; D. Dentine; D'. D'. Internal wall of dentine showing clefts probably due to molecular changes; P. Pulp tissue beginning to degenerate. Magnified 45 times.

The knowledge might perhaps explain the deaths of pulps where no evident lesion could be ascertained.

And, finally, it is not difficult to believe that, when the blood system of the pulp is deranged in the way already described, it is deprived of its trophic functions with regard to the tissues around. Teeth lose their accustomed vitality and become more susceptible to outside influences and disturbances. At first remaining *in situ*, though affected by fibrosis, which exists unknown both subjectively and objectively, the degenerated pulp may begin to transmit its lowered vitality to the dentine and the periodontal membrane. The former most probably is deprived of its protoplasmic nourishment, wholly or in part, and undergoes more or less chemical or molecular change, and at times morphological change too, as seen in the photomicrograph (Fig. 225), becoming unduly brittle. The latter may easily and doubtless does share in the fibroid degeneration, which would assuredly sooner or later tend to loosening of the teeth in their alveolar sockets. It may, in addition, be hinted that perhaps this condition of lowered vitality may predispose the teeth to the onset of dental caries.

CHAPTER X

THE MORBID AFFECTIONS OF THE ALVEOLO-DENTAL PERIOSTEUM

MICROSCOPICAL ELEMENTS IN:—(i) Inflammation; (ii) Abscess; (iii) Dental cyst; (iv) Innocent and Malignant tumours.

Inflammation

Etiology.—The causes may be divided into local and general. Among the first may be mentioned: (i) Extension of septic diseases from the pulp; (ii) Cold; (iii) Application of drugs or other irritants; (iv) Mechanical irritation of and infection around masses of calculus; (v) Traumatism, such as a blow, fracture, use of toothpick,

FIG. 226.—Maxillary canine with chronic inflammation of the periodontal membrane.

FIG. 227.—Maxillary premolar with chronic inflammation of the periodontal membrane.

etc., and (vi) Systemic. Amongst the latter: (i) Mercurial salivation; (ii) Systemic dyscrasia, *e.g.*, gout, rheumatism, syphilis, struma, etc., and rarely (iii) Phosphorous poisoning.

Synonyms.—Periodontitis, pericementitis, etc. It may be local or general; acute or chronic.

Secondary Changes.—Resolution, suppuration with sometimes necrosis and absorption of the alveolar bone.

Macroscopical Appearances.—The tissue is more or less thickened, rough, and blood-stained. The removal of the tooth imparts to it a ragged appearance. In places it may be stripped from off the cementum.

Symptoms.—When acute there is pain which is constant and dull in character, relieved in early stages by pressure on the affected tooth, but returning when the pressure is removed. The tooth becomes in time loosened; usually it returns to its normal firm condition. When chronic, pain is considerably modified; may even be absent until pressure is applied locally. The tooth is permanently loosened, and thus a great difference exists between the acute and chronic conditions.

DIFFERENTIAL DIAGNOSIS OF ACUTE INFLAMMATION OF:

The Pulp	The Periodontal Membrane
1. PAIN. Sharp, shooting, intermittent, throbbing, reflected.	1. Dull, gnawing, aching, continuous, localized.
2. TEMPERATURE. *A*. Cold may give relief in early stages. *B*. Heat intensifies pain.	2. *A*. Cold generally gives relief. *B* Heat does not alter character of pain.
3. INSPECTION. Tooth normal height.	3. Tooth raised in socket.
4. PALPATION. Tooth firm.	4. Tooth loose in later stages.
5. PERCUSSION. Negative.	5. Induces pain.
6. PRESSURE. Negative.	6. At first relieves pain; in later stages, intensifies it.
7. CAVITY. Generally present.	7. No cavity.
8. PAIN. Increased on assuming recumbent position.	8. Not increased.

HISTOLOGY

The fibrous tissue is infiltrated with inflammatory cells and products. These new cells collect in masses between the fibres, and are found running in a direction chiefly parallel with the axis of the root of the tooth. Inflammatory foci may form and suppurate, producing small localised abscesses. The osteoblasts are particularly visible, and depositions of new cementum with irregular lacunæ and canaliculi often occur. Calcospherite spherules may also sometimes be found. The blood-vessels are dilated, and the perivascular tissues with their nerve bundles are considerably increased in size, as well as in the number of their individual elements.

Abscess

HISTOLOGY

Suppurative foci of varying dimensions may occur in any part of the periodontal membrane. When of large size they are called alveolar abscesses. The inflammatory process has passed into the

stage of suppuration, and according to the rate of formation and the severity of the symptoms produces an acute or chronic abscess. In both cases, not only is the periodontal membrane affected, but the bony sockets also. When the inflammation becomes chronic, the membrane becomes extensively thicker, through the persistence of the changes occasioned by the acute condition. After extraction of a tooth thus affected, it is seen that large masses of soft tissue are adherent to the cementum. This soft tissue very frequently becomes transformed rapidly into granulation tissue, with all its char-

FIG. 228.—Acute inflammation of the periodontal membrane *in situ*. Prepared by the Author's process. Stained with hæmatoxylene. Magnified 40 times. D. Dentine; c. Cementum; P.M. Root membrane, the fibrous tissues of which are crowded with inflammatory cells and products.

acteristic appearances and consequences. The alveolar socket becomes absorbed, and if the process continues, the cementum in addition, by the functions of phagocytes. "Granulomata" have been described in this connection, but nothing warrants the application of this appellation to the condition, when the correct definition of a tumour is taken into consideration (see page 327).

Microscopical examination shows that the abscess consists of an outer firm fibrous sheath forming a kind of wall, the fibres of which run side by side in a longitudinal direction all closely interlacing. Stretching from these, inwards, are numbers of trabeculæ, composed of fine connective tissue dividing the interior of the cavity into
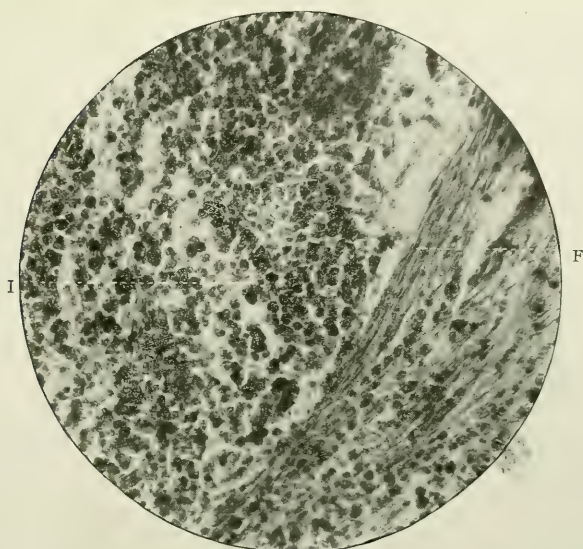
Fig. 229.—The same as the preceding.   Magnified 250 times.   F. Connective tissue ("principal") fibres; I. Inflammatory cells and products.
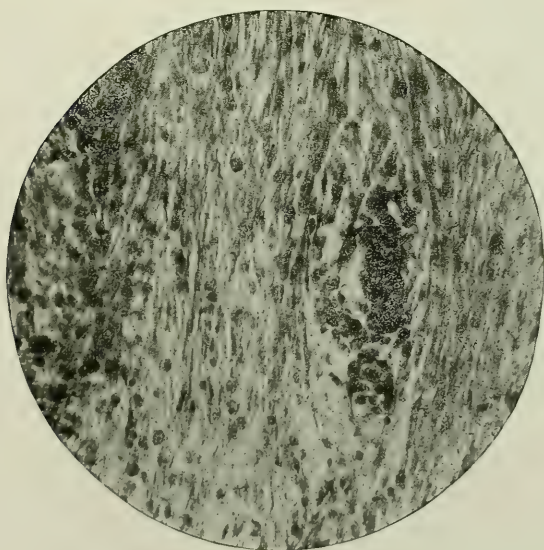
Fig. 230.—Another part of the same.   Magnified 250 times.

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septa or compartments of varying size and shape. Each is filled
with round or squarish cells of large dimensions. In the centre of
all, the majority of the pus cells have escaped during the extrac-
tion of the tooth, the force of which has ruptured the wall.

FIG. 231.—A section through a chronic abscess of the periodontal membrane.
Stained with hæmatoxylene. Magnified 50 times. c.t. Capsule of fibrous
tissue; M. Connective tissue cells; c. Cavity occupied by *liquor puris* and pus
corpuscles.

Dental Cyst

Definition.—A cystic degeneration of the epithelial "rests" of
the periodontal membrane, produced as a result of inflammation
of that tissue, containing some viscid fluid and holding crystals of
cholesterine and other salts in suspension, and lined with epithelium

Etiology.—Two hypotheses are prevalent as to its origin—(A) the mesodermic and (B) the ectodermic.

(A) The former is based on the belief that either (i) granulation tissue having attained a considerable size may break down, and caseate, or (ii) a chronic abscess may secrete a fluid. (B) The latter is the theory fully investigated and described by J. G. Turner,[1] who has conclusively proved that minute quantities of septic products from the pulp cavities of dead teeth, setting up a chronic inflammation of the periodontal membrane may stimulate to activity and rapid growth the paradental epithelial "rests" of Malassez ("débris épithéliaux paradentaires"). The central epithelial cells being cut off from their blood supply die, degenerate and liquefy, and a cyst results.

This is most probably the correct view.

FIG. 232.   FIG. 233.

FIG. 232.—Maxillary non-carious molar having a large dental cyst attached to the periodontal membrane of the buccal roots. It became displaced during the removal of the tooth, and, rupturing, its contents were evacuated. Mesial aspect.

FIG. 233.—Same as the preceding. Buccal aspect.

A dental cyst does not conform at all closely with the lines of classification enunciated by certain general pathologists. In the first stage of its evolution it is not derived from a distension of pre-existing cavities or spaces such as are a bursa, a ganglion, a ranula, a galactocele; it is not a cyst of new formation, like an adventitious bursa, a hæmatoma, a proliferous compound cyst, a parasitic (hydatid) or an implantation cyst; neither is it of congenital derivation, as is, for instance, a dermoid cyst or a cystic lymphangioma. The important point to emphasise is that it owes its

[1] "Dental Cysts," *Journal British Dental Association*, pp. 711, *et seq.*, October, 1898.

being to an infective condition of the tissue in which it is found, whereas none of the cysts enumerated above directly does.

It has recently been shown[1] that dental cysts are not entirely dependent on their origin on teeth with septic pulps, as in the instructive case referred to the pulp was alive. It was suggested that it was the outcome of spontaneous growth on the part of the same epithelial mass or masses. In the ensuing discussion, Mr. Turner expressed the opinion that it might have arisen in connection with a neighbouring tooth, which had been extracted fifteen years previously to the occurrence of the cyst. Incidentally he also mentioned that a cyst may originate as a consequence of inflammation of the periodontal membrane due to "pyorrhœa alveolaris."

General Characteristics.—For a classification and the differential diagnosis of cysts of the jaws, see Chap. XV, page 401.

Macroscopical Appearances.—On their facial aspects they present a smooth, oval, or round cystic tumour of different sizes according to the stage of growth, having a firm but elastic capsule. The contents may vary from a yellow thin liquid to a green pultaceous substance.

Secondary Changes.—Inflammation and suppuration; which under suitable conditions may lead to spontaneous cure. It is possible that carcinomatous degenerations may occur.

HISTOLOGY

A dental cyst, after evolving from a solid collection of epithelial cells, consists of a connective tissue capsule or wall of varying degrees of thickness,[2] which encloses masses of epithelium.

There is a small round-celled infiltration into the capsule, which is most pronounced near the epithelial lining. Russell's fuchsine bodies are sometimes present.

The epithelium may vary in character and amount. Thus the cells may be round or oval with flattened nuclei, or columnar with spherical nuclei, or even occasionally ciliated. The presence of cilia has been demonstrated by Turner, Baker[3] and Rothmann.

[1] Ernest B. Dowsett, "Dental Cyst arising from the root of a living tooth," *Trans. Odonto. Soc. of Great Britain*, Jan., 1901.

[2] In some specimens examined by the author it was 3.25 mm. in width, the oral mucous membrane being 0.9 mm. in width.

[3] "Notes on the Pathology of a Dentigerous Cyst," *Dublin Journal of Medical Science*, October, 1891.

FIG. 234.—Russell's fuchsine "bodies" in wall of a dental cyst. Stained with
hæmatoxylene and fuchsine. Magnified 250 times. B. "Bodies."
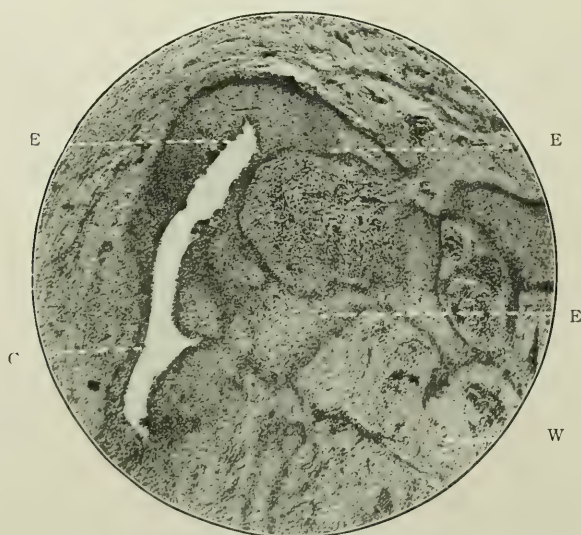
FIG. 235.—The interior of a small dental cyst. Stained with hæmatoxylene
and eosine. Magnified 45 times. C. Cystic cavity; E. Epithelial lining. W.
Cellular infiltration of fibrous tissue of wall of cyst.

As regards the occurrence of ciliated columnar epithelium in dental cysts, Turner thinks that it may be a "reversion to type or a freak of growth," while Baker writes as follows:—
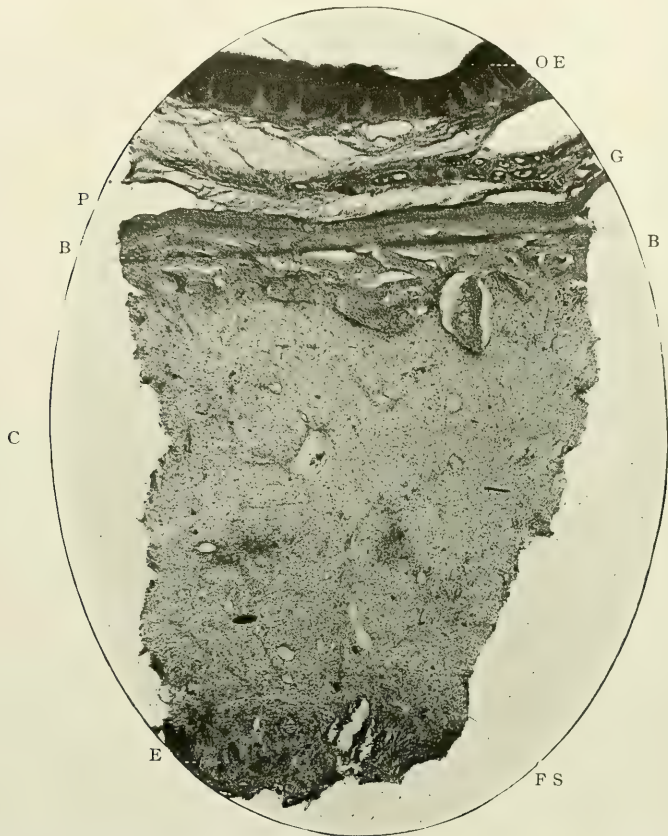
FIG. 236.—A section through the entire wall of a dental cyst. Prepared by fixing and hardening in formic aldehyde and alcohol, and carefully decalcifying in a weak solution of hydrochloric acid. Stained with Ehrlich's acid hæmatoxy-lene, counterstained with eosine. Magnified 20 times. o.e. Oral epithelium; G. Gum tissue; P. Periosteum slightly detached from surface of the bony wall; B. Attenuated bone; c.w. Wall of cyst; E. Epithelium; F.S. Inner surface of cyst wall.

"At first sight it seems a little difficult to account for the presence of columnar epithelium in a position remote from the respiratory tract and those other places where it is usually found: still if we remember the way in which ciliated epithelium is regenerated, its presence in my case will not be quite so obscure. It is a well-

FIG. 237.—A section through a small dental cyst. c.w. Cyst wall; c.e. Ciliated epithelial lining; c.c. Cyst cavity; o.s. External surface of the tumour; r. Extremity of root of tooth.   (*Prepared and photographed by A. W. W. Baker.*)

FIG. 238.—A portion of the preceding.   More highly magnified. c.e. Ciliated epithelium; c.i. Cellular infiltration; c.c. Cyst cavity.

known experiment that when the ciliated epithelium is artificially removed from a portion of the inner surface of a rabbit's trachea, the denuded surface speedily becomes again covered with epithelium, which grows over it from the edge.   But the cells form at first a single layer of flattened epithelium; they next acquire cilia, and afterwards become columnar, the epithelium thus assuming the character which it has normally in that situation. If such transformations are possible, there is no reason why the epithelium, which is frequently present in the root membrane, should not under suitable conditions become changed into columnar ciliated."

In amount the epithelium is sometimes merely a thin layer of flattened cells, sometimes a thick dense collection into which the sub-lying tissues send papillary eminences.

Cholesterin, which is a monatomic alcohol and soluble in ether, may appear in the fluid contents as numerous, flat, minute, rhomboidal crystals each possessing one broken corner.

Tumours

In addition to the cystic tumour already noticed, there occasionally occur homologous neoplasms which belong to the connective tissue and epithelial groups of tumours affecting soft tissues. This is not surprising when the histological characteristics of the root membrane are recollected.   Hence, the new growths, which are not due merely to hypertrophy of soft tissues or to inflammatory changes within, may be anatomically classified as:

    A. Connective tissue tumours—
        1. Tumours of the type of fully-formed connective tissues,
        2. Tumours of the type of the higher connective tissues,
        3. Tumours of the type of young connective tissue.
    B. Epithelial and glandular tumours.

Of these main divisions, the periodontal membrane possesses, as far as investigations go at present, representative growths belonging to the types of

      1. Fully-formed connective tissue, *e.g.* Fibromata,
      2. Young connective tissue, *e.g.* Sarcomata, and
      3. Epithelial tumours, *e.g.* Carcinomata.

1. *Fibroma*

The macroscopical appearances of this benign growth arising from the periodontal membrane are depicted in Figs. 239, 240, and 241.

A full description of these tumours will be found in Chapter XIII

FIG. 239.   FIG. 240.   FIG. 241.

FIG. 239.—Maxillary right first incisor with fibroma arising from the periodontal membrane. Distal aspect.

FIG. 240.—Same as preceding. Labial aspect.

FIG. 241.—Same as preceding. Mesial aspect.

2. *Sarcoma*

Oakley Coles, in 1885, was the first to draw attention to the existence of round-celled sarcoma of the root membrane, the diagnosis and microscopical examination having been undertaken by Klein.
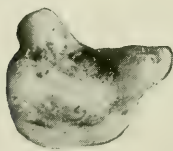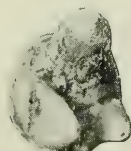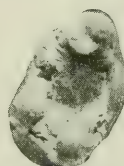
FIG. 242.   FIG. 243.   FIG. 244.

FIG. 242.—Round celled sarcoma of the periodontal membrane beginning between the roots of a non-carious maxillary molar. Radicular aspect.

FIG. 243.—Another non-carious maxillary molar similarly affected to that in preceding figure. A more advanced condition. Radicular aspect.

FIG. 244.—Another non-carious maxillary molar, similarly affected to that in preceding figure. A more advanced condition. Radicular aspect.

And the author has observed several possible cases. There is some shadow of doubt that all these instances are true sarcomata. The close approximation of appearances presented by inflammation of the root membrane, makes this element of uncertainty. But it

must be remembered that all fibrous tissues, especially those of the periosteum of bones, have a special predilection to undergo sarcomatous changes; and the clinical histories must not be ignored.

The chief points of interest in connection with these periodontal tumours lie in the facts that they are found in connection with the roots of sound teeth, and that their characteristics are those of round-celled (alveolar) sarcomata.

Seats of Occurrence.—In half-a-dozen cases which have come under the immediate observation of the author, each growth was confined, as its *locus principii*, to the periosteum of the molar teeth, the maxillary being much oftener affected than the mandibular series. It was generally seen to rise from a point situated at the junction of the roots with the body of the tooth; but it may have its origin from the sides of one or even two roots. Later, it generally fills up the whole of the interradicular region of the tooth (Fig. 244).

The *Etiology* of the disease is obscure; but there seems to be a predisposition on the part of the growths to attack the fibrous membranes of the teeth of women about the period of the menopause. Long-continued and powerful friction, as shown by the wearing down of the cusps, is possibly the exciting cause.

The *Subjective symptoms* point chiefly to long-continued sharp pain, increased on pressure, the course of the disease lasting sometimes several months. The pain is severe at times, and such as to render necessary immediate extraction of the loosened organ.

Objective Symptoms.—On examining the mouth, at first there is sometimes almost entire absence of swelling or of any of the usual inflammatory signs, and the tissues are not markedly indurated. There may be slight suppuration. If the disease is not far advanced, diagnosis is only complete after the removal of the tooth. Later, well-marked symptoms of malignancy appear.

The tumours vary in size from a split pea to a small nut, and have a smooth, convoluted, rarely ragged, surface. They are firm to the touch, and are of a deep red colour. The teeth themselves are non-carious, and exhibit in their hard parts no traces of disease except some attrition of their cusps and (in some cases) absorption of the apices of the roots. They are markedly loose, and signs of chronic inflammation of the periosteum, accompanied by an accumulation of tartar, are often noticed.

HISTOLOGY

The growths consist of masses of cells held together by a fine network of fibrous tissues which is very dense here or very loose there, and is in some places apparently undergoing fibrification or calcification.   In the centre of the growth this network is scanty, but the intercellular tissue is conspicuous outside.   Vessels are scanty in the centre and have extremely thin walls; they ramify among the cells.   In the outer portion they are larger (but not dilated) and have normal walls.   The cells themselves are for the
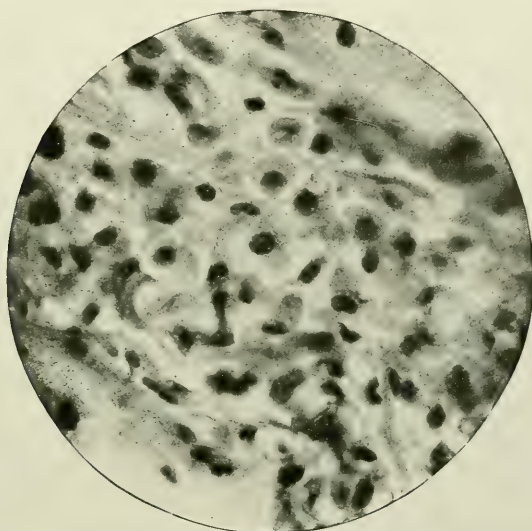
FIG. 245.—A small round-celled sarcoma of the periodontal membrane.   Stained with hæmatoxylene.   Magnified 750 times.

most part rounded in shape and considerably larger than red blood corpuscles.   They contain one or more nuclei and are devoid of any definite cell wall (see Fig. 245).   Great numbers of spindle cells exist.   There is little hæmorrhage into the tissues, probably because of the small size of the growth, and because it has not advanced sufficiently to allow of large hæmorrhages to take place in its substance; but small extravasations of blood corpuscles are noticed here and there.   Microscopically the growth is practically indistinguishable from granulation tissue; considered from a clinical aspect, however, there can be no doubt as to its malignant nature.   A jaw, excised for malignant disease of the antrum, which

is now in the possession of the author shows unmistakable signs of the sarcomatous growth beginning in the root membranes of the premolar and molar series, and extending into the floor of the antrum, and surrounding alveolar portions. It infected the soft tissues very thoroughly. This specimen for which the author is indebted to his friend the late Mr. W. J. Pilcher, was believed by this surgeon to originate *de novo* in the root membranes of the teeth of the patient, who was a young male adult of about 18 or 20 years.

To sum up, it may be said that sarcomatous disease of the periodontal membrane is not rare in its earlier forms, but that it is very seldom met with in an advanced condition; and that removal of the affected tooth fortunately cuts short its career if taken sufficiently early, but if it is allowed to continue, it constitutes another starting-place for malignant disease of the maxillæ.

Less malignant, but very rare also, are tumours occasionally arising from the periodontal membrane, which exhibit the characteristic giant-cells of myeloid sarcoma.

They will be more fully described in Chapter XIII; but here it may be said that while they present on the gum surface as a so-called myeloid "epulis," they have sometimes distinct connections with the periosteum of the roots of teeth.

3. *Squamous celled carcinoma*

Epithelial malignant tumours of the alveolo-dental membrane are very rare. But their rarity in no way detracts from the importance of their early diagnosis, and of the strictest prophylactic measures to be always taken with regard to their possible source of future development.

In the *Trans. Odonto. Soc. of Great Britain*, June, 1901, and under the title of "A Case of Burrowing Epithelioma," Dr. Stanley Colyer has succinctly described a remarkable illustration of this affection. A similar case of "boring" epithelioma ("burrowing carcinoma") had previously been described by Sir Henry Butlin (*Pathological Trans.* vol. xxxii, 212, 1881), while Reclus, in 1876, was the first to note and record the condition. This is not the place for full references to be made to these communications. The reader is referred to the pages of the Transactions (pp. 231 to 242), for Dr. Colyer's statements.

Nevertheless the writer here quoted may be allowed to express his opinion as to the possible and probable etiology of cancer of the

periodontal membrane, an opinion which, it may be added, is completely endorsed by the author.

Etiology.—The pathological history of the case was somewhat as follows:—

"The pulp was infected through a carious cavity and died; it then set up a suppurative inflammation around the apex. The pus pointed and left behind it an incurable sinus which would suggest —at any rate after the tooth had been sterilised—that some portion of the tooth was necrosed. The necrosed portion, acting as an irritant, would cause a hypertrophy of the stripped-up pericementum and a proliferation of the epithelium therein. The pain, though not great, would put the tooth out of use, and the pericementum,

FIG. 246.   FIG. 247.   FIG. 248.   FIG. 249.

FIG. 246.—Squamous celled carcinoma arising from the periodontal membrane of a maxillary left first incisor. The apical portion of the root was much absorbed. Labial aspect.

FIG. 247.—Squamous celled carcinoma arising from the periodontal membrane of a maxillary right first incisor. Labial aspect.

FIG. 248.—Squamous celled carcinoma arising from the periodontal membrane of a maxillary left canine. Apical portion of the root considerably absorbed. Distal aspect.

FIG. 249.—Squamous celled carcinoma arising from the periodontal membrane of a maxillary left premolar. Mesial aspect.

which . . was not thickened, would be functionally isolated. We have, therefore, got a functionally isolated pericementum in an otherwise healthy body, a condition which is analogous to the breast and uterus about the time of the menopause. According to this theory, then, the nice balance existing between the cells of the pericementum is lost, the epithelium reverts to its ancestral powers of division, and burrows into the surrounding tissues, whose physiological resistance is reduced; and thus the cancer is born."

Macroscopical Appearances.—There is practically nothing to assist the diagnosis of the lesion by gross examination. A soft mass of firm consistency arising from the periodontal membrane of a "dead" tooth, with or without disease of the cementum and dentine may be

chronic inflammation, a dental cyst (in incipient stages), a localised and circumscribed abscess, a round-celled sarcoma, or an epithelioma. Histology not only helps the differential diagnosis, but serves again, in a striking manner, to emphasise the dangers attendant on the killing of pulps in teeth, and the subsequent insufficient cleansing of the pulp canals. It must never be forgotten that "dead" teeth, unless they have undergone a rigorous course of scientific treatment, may seriously menace the health, or even bring about the death of patients.
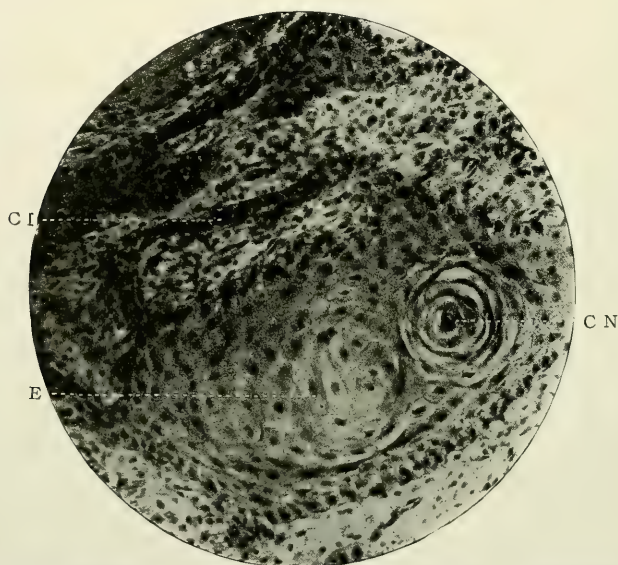
FIG. 250.—Epithelioma of the periodontal membrane ("Burrowing epithelioma"). Stained with Ehrlich's acid hæmatoxylene. Magnified 230 times. c.n. Cell-nest; c.i. Small celled infiltration of the tissues; e. Island of epithelial cells.

HISTOLOGY

As far as the microscopical appearances of the specimens under consideration go, it is unnecessary to detail the various structures which comprise the substance of the growth. Epitheliomata agree in all essential particulars wherever found. For a description of carcinomata of the gums and palate, the reader is referred to Chapter XIII. The appended photomicrograph (Fig. 250) gives the general appearances of such a specimen.

"PYORRHŒA ALVEOLARIS"

Introductory—The gingival margin—The periodontal membrane—The apical region—The cementum—The bone of the jaw—Conclusions—Normal arrangements of the osseous and fibrous tissues—Early changes producing osseous atrophy—Absorption by granulation tissue—Chronic periostitis and senile changes—"Pockets"—Anatomical and clinical observations—Summary.

INTRODUCTORY

The oral diseases of which "pyorrhœa alveolaris" is a symptom have engaged the attention of many observers from the days of Fauchard in 1746 and Jourdain in 1778 to the present time, when, more than ever, the whole of the medical profession, as well as the modern dental surgeon, has become acquainted with them in a greater or less degree. Next to dental caries, "pyorrhœa alveolaris" has especially attracted the notice of dental surgeons on account of its universality, its controversial character, its obscure etiology, its occasional difficulty of diagnosis, its recondite morbid anatomy, its usual intractability to treatment. It would be futile and distinctly beyond the scope of this chapter to review, however briefly, the whole field of literature relating to it. In spite of all that has appeared it is still uncertain as to whether the flowing of pus from the alveolar sockets of the teeth is a local or a constitutional symptom, or both.

Definition.—Literally—"A flowing of alveolar pus." It is a symptom or sign of an infective condition of the socket or sockets of the teeth. Purulent effusion may be limited to one tooth or may be general. It may exist for years localised, or rapidly spread. It is accompanied by a slow wasting or atrophy of the alveolar processes of the jaws, and may be attended by the formation of tartar, and gradual and progressive loosening of the affected teeth. It is generally painless, and is not necessarily inflammatory in origin or in consequence. Chronic inflammations of the periodontal membrane frequently exist without "pyorrhœa." *Synonyms* are

numerous: "Rigg's disease," "interstitial gingivitis," "hæmato-genic calcic pericementitis," so-called "periodontal disease," etc.

Etiology.—Undetermined at present: but probably constitutional diseases, coupled with infection of the gum margins with pyogenic cocci, may briefly be considered potent factors in its causation. Three great schools of thought hold conflicting views on this much-debated question. A discussion of these would not be germane to the subject-matter of this book. Suffice it to say that the following, in a word, represent these conceptions:—

(i) It is occasioned solely by local conditions setting up inflammation of the gums, through the deposit of tartar, etc. A belief of Riggs (*Trans. American Academy*, 1875), Witzel (*British Journal of Dental Science*, 1882), Black ("American System of Dentistry," 1886),

(ii) It is due to bacterial infection—opinions of Arkövy ("Diagnostik der Zahnkrankheiten," S. 232, 1885), Galippe ("Die infectiose arthro-dentare gingivitis," 1888), and Miller ("Microorganisms of the Human Mouth," 1890), who found twenty-two varieties of bacteria in twenty-seven different cases,

(iii) And finally, it has been ascribed to a constitutional origin such as gout, rheumatism, etc., theories shared alike by Tomes (*op. cit.*), Fitzgerald (*Clinical Journal*, 1899), Pierce (*International Dental Journal*, 1892, 1894, and 1895), and many others including Kirk, Burchard, and Talbot ("*Interstitial Gingivitis*").

Znamensky of the University of Moscow, with the assistance of Dr. Saricheff and Professor Nikiforoff, published some years ago a most careful paper on this subject. He was able to give an account of the microscopical appearances of the diseases associated with "pyorrhœa alveolaris," for he had obtained sections through the teeth and jaws of a woman suffering from alveolar "pyorrhœa," but who had died from another disease. This is described in the later portions of this chapter.

The author has had the opportunity of making sections through the dental and alveolar tissues of several patients who had "pyorrhœa alveolaris."[1] In this chapter he proposes to put on record a description of this material, with the conclusions derived from a preliminary study of them.

The first specimen was the right maxilla from the mouth of a male twenty-eight years of age. Vertical cuts were made with a

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[1] The jaws were incised after death, of which the local conditions in the mouth were no contributory cause.

diamond disk through the canine and molar teeth and an interdental septum, the roots of the molar being largely exposed on both the
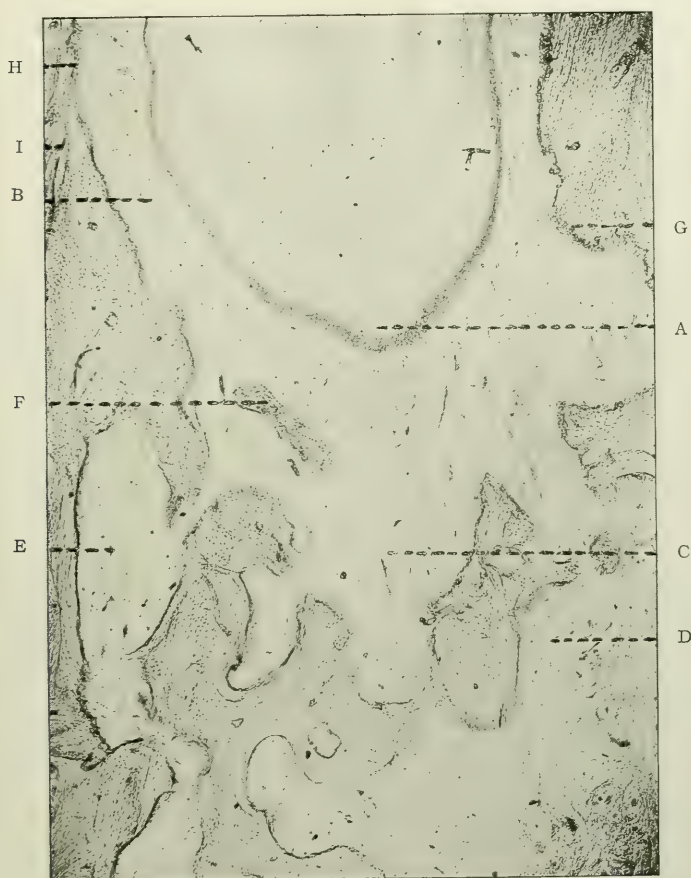
Fig. 251.—Vertical section through canine and right maxilla of man aged twenty-eight showing latest stages of extremely acute conditions associated with "pyorrhœa alveolaris." Lateral section. A. Apex of root formed by hyperplasic cementum; B. Hyperplasic periodontal membrane; C. Indifferent tissue enormously increased in amount and more vascular than usual; D. Soft medullary tissue exhibiting signs of hyperplasia; E. Large osteoporotic space; F. Sequestrum of bone undergoing peripheral absorption; G. Osteoclasts producing lacunar absorption of bone of socket; H. Bone of socket partially destroyed and converted into osteoid tissue; I. Line of junction of decalcified and normal bone. Magnified 35 times.

labial and lingual sides. Macroscopic examination of these cuts showed that the gingival margin in the canine and first premolar

region was greyish in colour, believed to be due partly to blood-clot
and partly to the presence of gangrenous tissue. The teeth were
sound, but were slightly loose. At the deepest part of the greyish
area a somewhat reddish line was observed, forming the border of
demarcation between the presumably healthy bone of the jaw and
the diseased superficial tissues. The first of these pieces was rapidly
decalcified by immersion in *aqua regia;* the latter were treated by the
Koch-Weil method of Canada balsam impregnation.
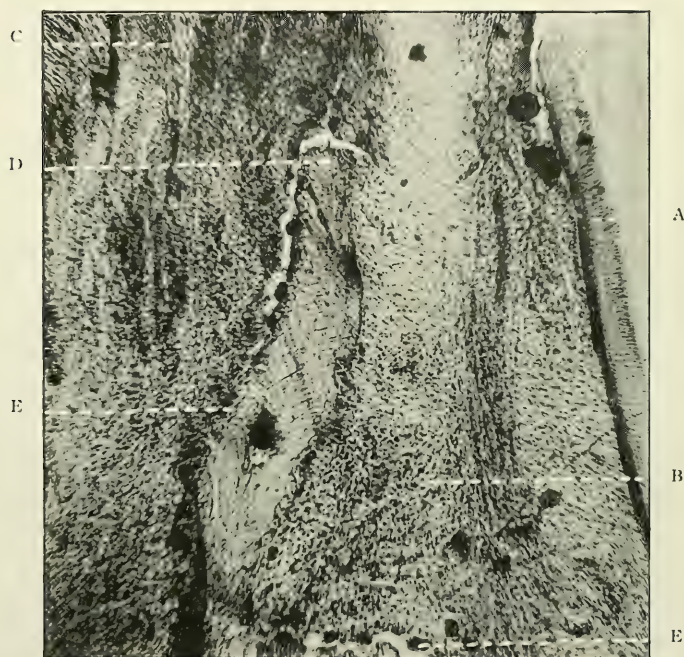
FIG. 252.—Free margin of edge of socket of labial surface of same. A.
Cementum; B. Inflamed periodontal membrane; C. Inflamed gum tissue; D. Free
edge of bone decalcified and converted into fibrous intervening tissue; E. Osteo-
clasts producing lacunar absorption of external and internal surfaces of bone.
Magnified 80 times.

The second specimen was a portion of the right side of the man-
dible of a patient of thirty-nine years, extending from the canine
to the second molar inclusive. The second maxillary premolar and
first molar had been previously extracted. Two vertical incisions
were made through the canine and bone, and one on the mesial
surface of the root of the second molar. The canine was found to
have undergone attrition and was non-carious. A slight band of

tartar was seen on the lingual surface. This was treated by the Koch-Weil process. The mass, including the molar root, was decalcified.

Another specimen consisted of the right maxilla of a patient of sixty years, from the canine to the second premolar inclusive. Two cuts were made through the canine. There was much attrition of this tooth, but no caries. Tartar was present on the labial surface. One piece was decalcified, the other hardened, after staining *en masse*, in balsam. The decalcified sections were cut on an ether-freezing microtome and stained with Ehrlich's acid hæmatoxylene and counter-stained with an alcoholic solution of eosine and mounted in Farrant's medium, as less likely to cause contraction of the soft parts. Some sections were stained by Gram's method for bacteria. The Koch-Weil specimens were stained with Grenacher's alcoholic borax-carmine.

More material was examined, but it is unnecessary to give further details.

The following description is the result of the microscopical examination of tissues profoundly affected by acute, later phases of the condition.

HISTOLOGY

From an anatomical point of view this may be conveniently considered under the following headings: (I) The gingival margins. (II) The periodontal membrane. (III) The apical region. (IV) The cementum. (V) The bone of the jaw. This is of course an arbitrary distinction, but it serves to simplify the descriptions of the parts.

The Gingival Margins

The oral epithelium is apparently altered in all parts, most especially at the periphery; here it has been lost, desquamation having taken place. Nearest the free edge, the cells of the *stratum corneum* and *lucidum* have undergone hydropic degeneration. Their nuclei have shrunken and many are moon-shaped in outline. In places where the epithelium has disappeared the papillary eminences of the corium remain as finger-like processes with free margins. Throughout there is a tendency for the epithelial cells to become confluent the "spiny" cells so commonly found in normal tissue being absent or indistinguishable. The deeper epithelial cells possess small round nuclei and are greatly increased in number, showing the phenomena of karyokinesis very markedly.

The submucous tissue, consisting usually of connective tissue fibres, is everywhere permeated by a round-celled infiltration, as the initial stage of an inflammation. This infiltration is most pronounced in the papillæ of that part of the gum which is most closely associated with the necks of the teeth. A "pocket" has already been formed. The capillaries are hyperæmic and the ducts
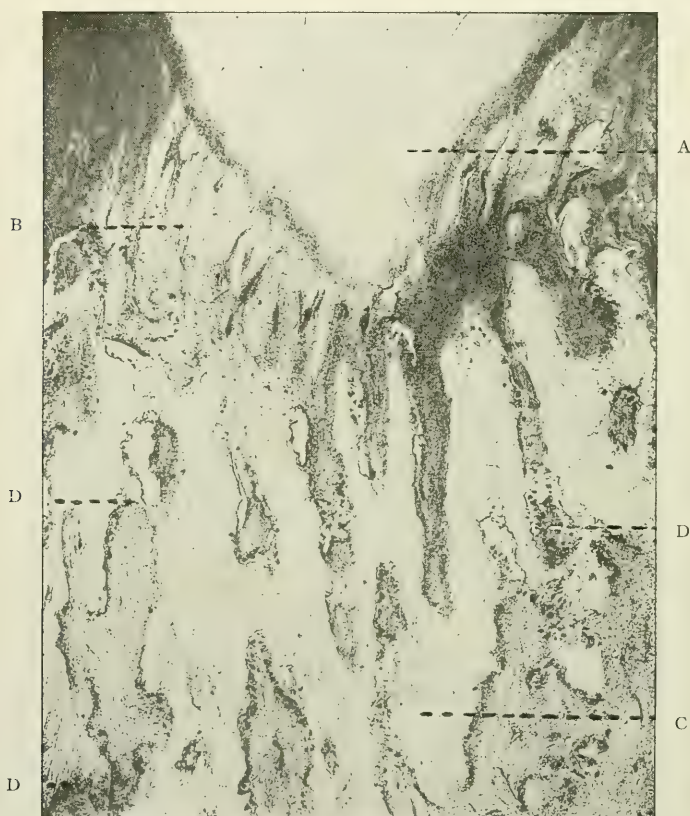
Fig. 253.—Apical region of same; median section. A. Apex of root with hyperplasia of cementum; B. Hyperplasic periodontal membrane; C. Alveolar bone, with many osteoporotic spaces; D. Osteoclasts. Magnified 35 times.

of the gingival glands appear swollen and more prominent than usual. The great masses of inflammatory cells consist of crowds of polymorpho-nuclear neutrophiles, eosinophiles, lymphocytes, and lymphoid cells, with numerous mast cells and Unna's plasma cells. They are congregated into groups or clusters between the fibres

of the gum tissue and of the periodontal membrane, which are all somewhat coarser than usual.

As the condition advances with the loss of the epithelium, the pathological changes are exaggerated to such a degree that dis-
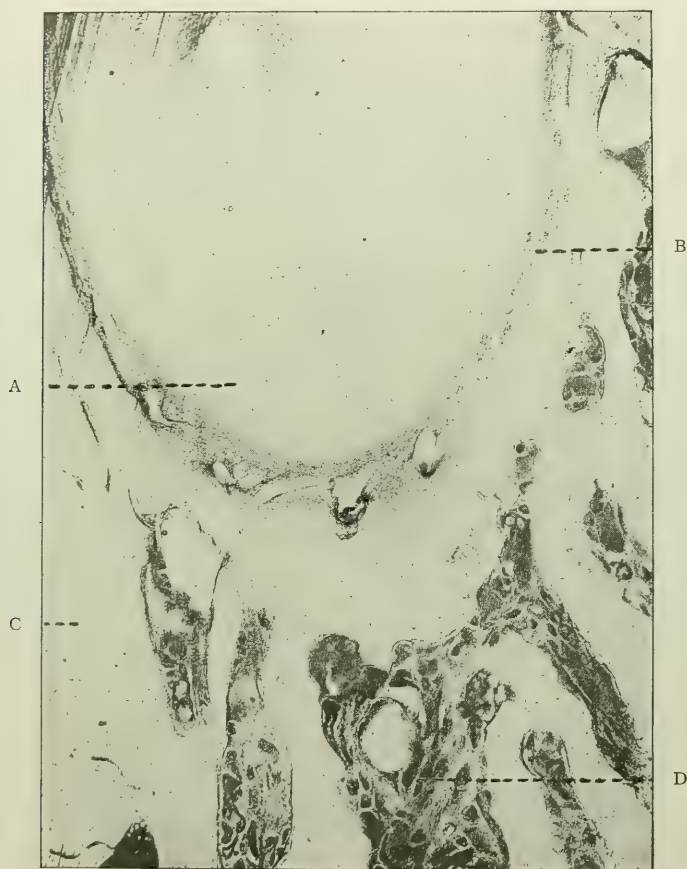
FIG. 254.—Vertical section through second incisor and left maxilla of woman aged twenty-five. Patient unaffected by disease of the bones. No "pyorrhœa alveolaris" present. For purposes of comparison with Fig. 253. A. Apex of normal root; B. Normal periodontal membrane; C. Normal alveolar bone; D. Normal medullary spaces and tissue. Magnified 35 times.

integration and death of cells and blood corpuscles has taken place, pus cells abound, coagulation necrosis and fatty necrobiosis of superficial tissues are noted, degenerative lipogenesis has occurred, and the main mass has become necrotic and friable, and is undergoing,

most superficially, liquefaction, due most probably to the proteo-
lytic ferments as well as to the enzymes of the bacteria.   Huge
colonies of micro-organisms penetrate the tissues from the surface
and can be seen by staining with gentian aniline violet—some of the
masses, however, resisting the colouration and appearing under the
16 mm. objective as deeply pigmented spots formed at the free

Fig. 255.—Vertical section through another part of canine of man aged
twenty-eight, showing destruction of socket. A. Dentine of root; B. Pulp canal;
C. Hyperplasic cementum at apex; D. Hyperplasic periodontal membrane; E.
Inflamed periodontal membrane; F. Sequestrum; G. Bone of external alveolar
plate; H. Slightly inflamed gum tissues; I. Bone of internal alveolar plate; J.
Osteoclasts producing lacunar absorption; K. Osteoporotic space; L. Osteoclasts
producing absorption of apex of root; M. "Gitter-figuren;" N. Thrombosed vessels
in periodontal membrane; O. Decalcified surface of G.   Magnified 35 times.

edge of the tissues.   The inflammatory cells of the early stages have
become so enormously multiplied that the nuclei are small and round,
and soon, at the more peripheral parts, pass insensibly into the
necrotic tissue just mentioned, which stains exceedingly feebly
and almost possesses a ground-glass-like appearance, traces of any
structure being absent.

But these intense changes are secondary to, and not the cause of
the "pyorrhœa alveolaris."

The Periodontal Membrane

(a) At the gingival region: Hyperæmia, cellular infiltration, hyperplasia of the fibrous elements, increase in diameter of the membrane—these are the most prominent features.

Normally measuring, in middle age, from 0.2 to 0.3 mm. in width, here it may extend to the enormous width of 1.0 mm., or at least

H   J
    E
    D
G
O
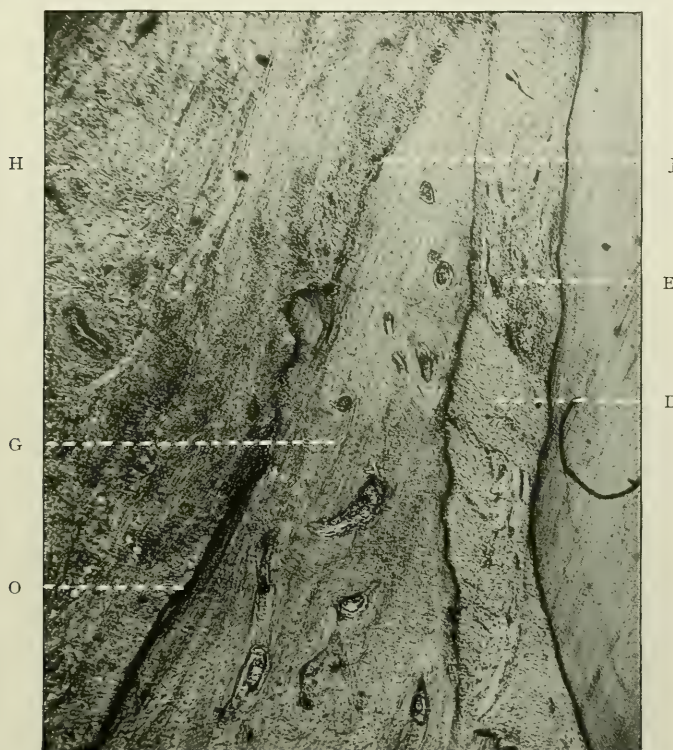
Fig. 256.—Portion of same.   Lettering as in preceding figure.
Magnified 30 times.

0.8 or 0.9 mm., the average width below being probably about 0.6 mm.   The increase in thickness is obviously due to the absorption of the edge of the alveolar process, which normally extends, as Znamensky says, "like a long narrow plate not containing any bonemarrow," of "the thickness of a piece of paper."

(b) Lower down, that is at the cervical region, the tissue shows signs of a slight increase in its cellular elements by proliferation of

the connective tissue cells. There is no great amount of round-cell infiltration or leucocytic invasion. In places it is not greatly hyperæmic and the tissue fibres are not well marked. Gland-like bodies are easily seen, but are not enormous nor important. But
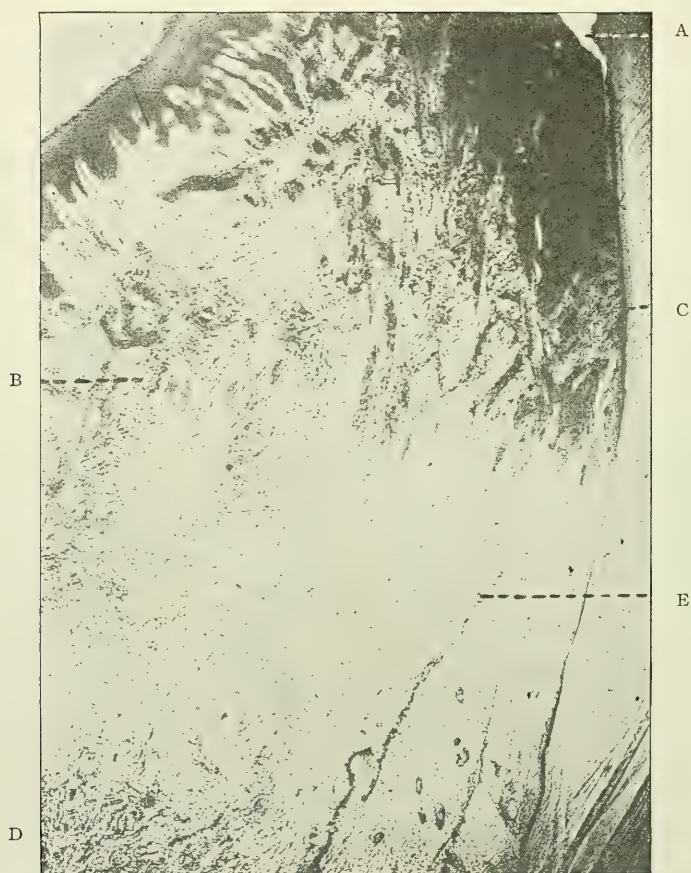
FIG. 257.—Gum tissue over external alveolar plate. A. Slight depth of "pocket"; B. Gum tissue slightly inflamed; C. Cementum; D. Normal gum tissue; E. Osteoclasts on surface of external alveolar plate. Magnified 80 times.

probably, taking it on the whole, it would be right to assume that there is a slight congestion of the soft parts, the main features of which are masked here by the strong fibrous elements. It never amounts to a general periostitis (periodontitis), and *there is no granulation tissue present except in very advanced conditions.*

The Apical Region

This, which usually measures 0.5 mm. in similar specimens, is now twice that thickness, filled with loosely arranged connective tissue fibres and the "indifferent tissue" of Black. Cells and blood-vessels (more numerous than normally), are increased in numbers and very prominent in the sections. The latter frequently branch and are filled with small cells.
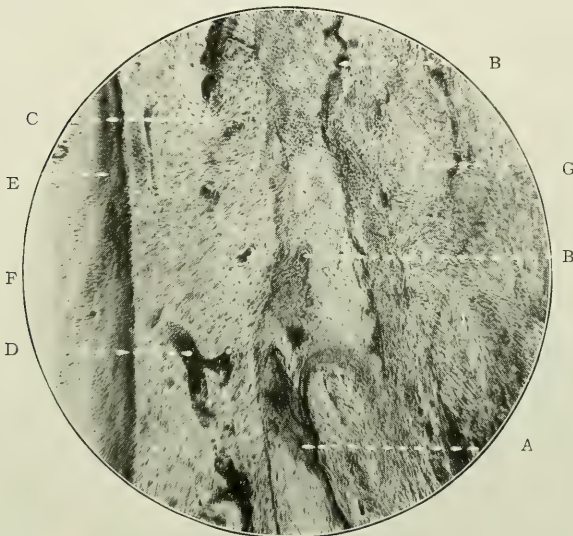
Fig. 258.—Lacunar absorption of the alveolar bone in "pyorrhœa alveolaris." Stained with hæmatoxylene. A. External alveolar plate which has almost become entirely absorbed; B. Osteoclasts on surface of A; C. Hyperplasic periodontal membrane; D. Thrombosed blood-vessel; E. Normal cementum; F. Dentine; G. Hyperplasic gingival tissue. Magnified 90 times.

The Cementum

The cementum, generally speaking, is hyperplasic, but not markedly so. Singularly enough its peripheral portions are remarkably smooth and well defined, and seldom exhibit, except at the apex of the teeth, the foveolæ of Howship occupied by large myeloplaxes. If it is at all hyperplasic it is accidentally so, due to some cause unassociated with the "pyorrhœa alveolaris."

There are no traces of bacterial invasion of Sharpey's fibres and their canals.

The Bone of the Jaw

The alveolar bone exhibits even before the gum the most important metamorphoses which have occurred. Briefly they are those produced by the process of halisteresis (perhaps a form of

FIG. 259.—Free margin of edge of internal alveolar plate. A. Cementum; B. Inflamed periodontal membrane; C. Bone; D. Osteoporotic space with inflamed medullary tissue; E. Osteoclasts; F. Inflamed gum tissue; G. Masses of micro-organisms. Magnified 35 times.

osteomalacia). Not only is the free margin of the bony socket absorbed by osteoclasts near the upper and lower parts of the cervical regions of the teeth, but deep down at the radicular portion, giving the surface an eroded appearance; moreover, there is also a

decalcification of the most superficial portions. The bone becomes transformed into an osteoid tissue through loss of its calcium salts, then passes into an intervening fibrous tissue and finally is attacked by the inflammatory exudation and cells, its bays and recesses becoming meanwhile greatly enlarged and filled with loose soft tissue, and the Haversian canals and medullary spaces enlarged and irregular, the condition being termed osteoporosis.

Absorption of bone may occur in three ways: First, by the activities of the osteoclasts of Kölliker (myeloplaxes); second, by the
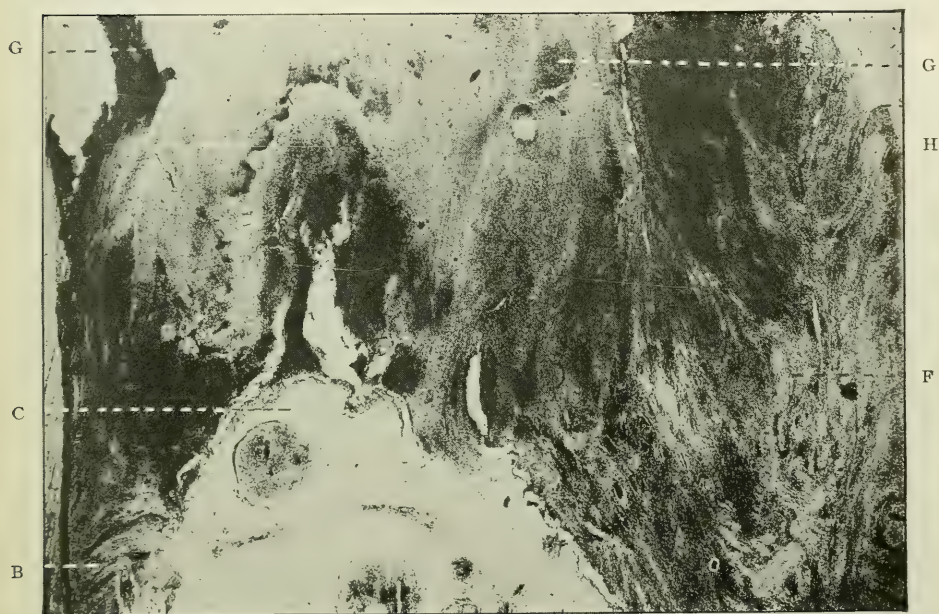
FIG. 260.—Another portion of same. Lettering as in preceding figure. H. Free surface of gum from which the oral epithelium has become desquamated.

process of halisteresis; third, by means of perforating canals. In the case of diseases such as give rise to the pathological conditions here studied, the two former are plainly going on side by side. The first is well understood. The second process may be described as being one of decalcification, the osseous matrix appearing as osteoid tissue. As the morbid changes progress, the osteoid tissue becomes more fibrillated than usual, and finally is dissolved and incorporated in the medullary tissue. Frequently are seen the *Gitter-figuren* of Von Recklinghausen, viz., variously shaped lines or markings in

the bone, depending upon its dissolution by the decalcific:tion agents and indicating its histodialysis (Fig. 261).

It is truly remarkable that the alveolar portions of the periodontal membrane exhibit numberless osteoclasts while the cemental portion does not. It is therefore probable that this portion of the membrane corresponds to the inner layer of the periosteum of bones generally—the proliferating layer of Virchow or the *cambrium* of
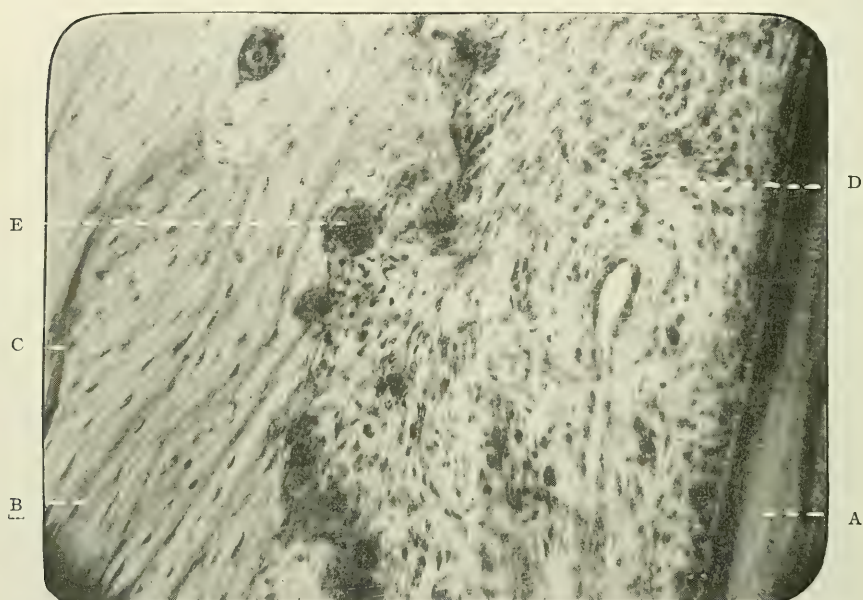
FIG. 261.—Another portion of same. A. Cementum; B. Bone; c. "Gitterfiguren;" D. Inflamed periodontal membrane; E. Osteoclasts. Magnified 250 times.

Billroth. The medullary spaces and Haversian canals throughout the sockets of the teeth are osteoporous—that is, have become eccentrically atrophic—a retrogressive phenomenon of no uncertain character. The same changes are occurring in the environment of the periodontal membrane, but not to so large an extent.

CONCLUSIONS

It is obvious from what has been said that the subject is vast, that the etiology of the flowing of pus from the sockets of the teeth is very debatable, and its pathology little understood. The author

ventures to state, based on his observations of the microscopical appearance of sections of teeth cut *in situ* from jaws which were distinctly pyorrhœic, that—

FIG. 262.—Vertical section through canine and bone of right side of mandible of man aged thirty-nine. "Pyorrhœa alveolaris" not marked. Tissues over external aspect. A. Dentine; B. Cementum; C. "Pocket" of gum, the edge of which is attached to the margin of cementum; D. Slight desquamation of oral epithelium; E. Slight inflammation of gum; F. Edge of alveolar bone transformed into osteoid tissue; bone lacunæ and corpuscles indistinguishable; G. Hyperplasic periodontal membrane; blood-vessels injected; H. Normal bone. Magnified 35 times.

(1) The disease of the bone is not in its earlier stages a rarefying osteitis. According to the most eminent authorities *osteitis rare: faciens* is "characterized by the development of vascular granulation

tissue of the medullary spaces and canals, associated with a lacunar absorption and canalisation of bone, which becomes porotic and soft." (Hektoen and Riesman, "A Text-book of Pathology,"
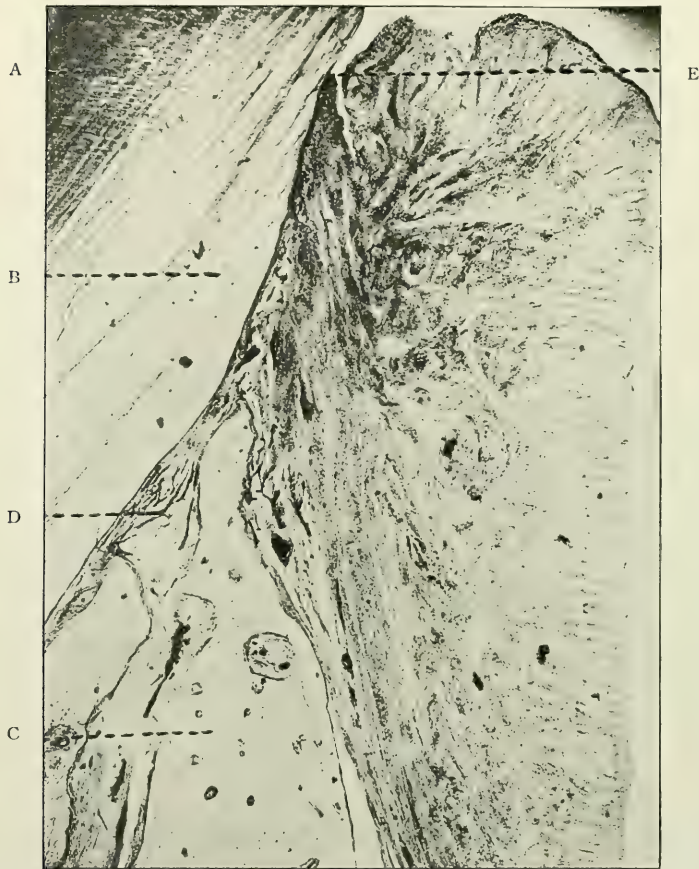
FIG. 263.—Vertical section through canine and socket of right maxilla of man aged sixty. Tissues over internal aspect. A. Dentine; B. Hyperplasic cementum; C. Bone of jaw; no lacunar absorption, slight decalcification of free edges; D. Periodontal membrane hyperæmic and showing signs of senile changes; E. Slight "pocket" at gum margin, slight gingivitis.

1901; see also "A Manual of Pathology," Joseph Coats, 1900; "A System of Surgery," Sir Frederick Treves, 1895; article by H. H. Clutton, "Fungating Ostitis (*Ostitis carnosa vel fungosa*)," Rind-fleisch's "Manual of Pathological Histology," 1873; and "Rarefying

Osteitis," in "An Introduction to Pathology and Morbid Anatomy," Henry Green, 1895.)

(2) "Pyorrhœa alveolaris" does not begin as a gingivitis.
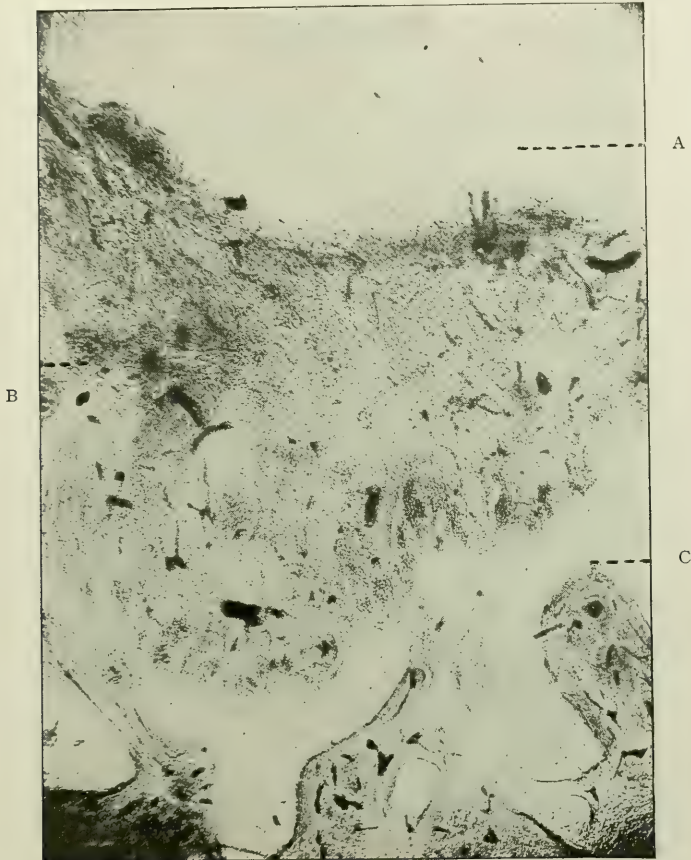
FIG. 264.—Same as before. Apical region of root. A. Dentine of root; B. Indifferent tissue enormously hyperplasic; C. Osteoporotic bone. It will be noticed that there is no osteoporosis at present at cervical region. (See preceding figure.)

(3) It is essentially dependent upon an osseous lesion, *an atrophy of the bone*, which, in the thinnest parts, causes the cervical margins of the teeth to become denuded through the halisteresis and osteoclastic absorption.

(4) The pus associated with it is derived from débris of food,

pyogenic bacteria, and other extraneous elements, which, gathering in the wide pockets produced by the atrophy of the bone, may or may not set up ultimately a suppurative gingivitis.
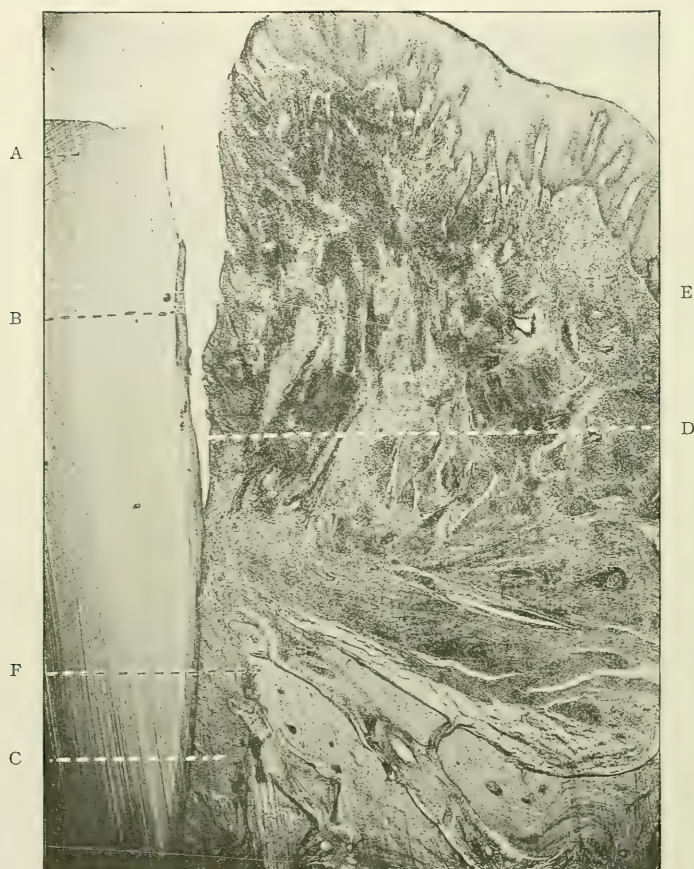
FIG. 265.—Vertical section through cervical region of molar of man, age unknown, which presented no symptoms of "pyorrhœa alveolaris." For purposes of comparison with the foregoing, and to demonstrate the fact that decalcification of edge of alveolar bone may exist before "pyorrhœa" sets in. A. Dentine; B. Cementum; C. Hyperplasic periodontal membrane; D. Gum tissue with deep gingival trough (nopus present); E. Normal gum; F. Decalcification of socket, no lacunar absorption. Magnified 35 times.

(5) The presence of calculus is not sufficient in itself to induce the condition, and may not be associated with it at all as a predisposing or exciting cause.

(6) Many cases of pus flowing from the sockets of teeth are not

"pyorrhœa alveolaris," but are frequently incorrectly described as such.

With regard to the statement made under clause (3) it seems impossible to imagine that if the gingivitis was, alone or combined with tartar, responsible for the pathological conditions, there could be no alteration in the bone *at the apices of the roots*, or in *situations far removed* from the surface of the gum. But this occurs; and it is
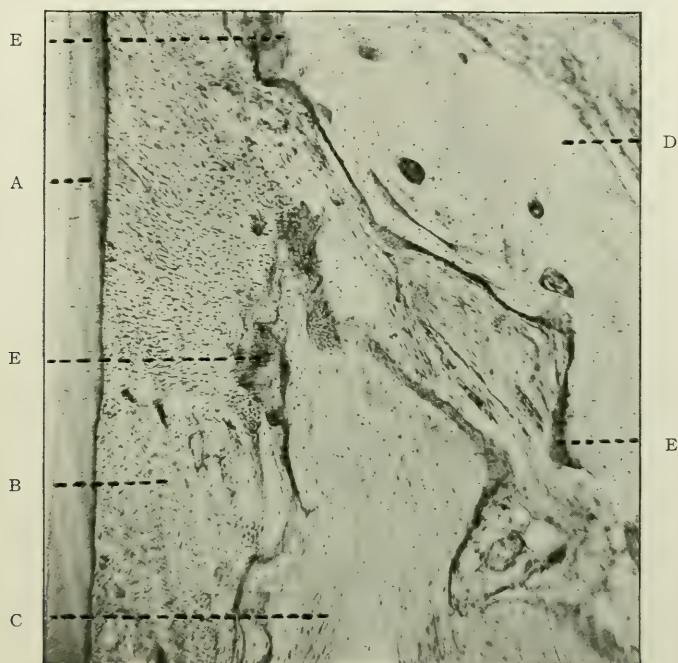
FIG. 266.—The same; shows tissues over internal alveolar plate. A. Cementum; B. Normal periodontal membrane; C. "Gitter-figuren;" D. Normal bone; E. Areas of decalcification. Magnified 250 times.

believed that the gingivitis, if present, is secondarily induced, and that this gingivitis is produced apart from the bony lesion by hæmatogenous infection by pus-producing micro-organisms, which, however, have a very circumscribed area of development.

NORMAL ARRANGEMENTS OF THE OSSEOUS AND FIBROUS TISSUES

Examination of sections of the teeth *in situ* in the jaws of a woman of twenty-five years, whose mouth was free from any symptoms of

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disease, discloses the fact (Figs. 267 and 268) that the alveolar proc-
esses vary in thickness, the bone being very narrow at the gingival
region, broader at the cervical region, while narrowing down again
at the radicular portion of the teeth to, roughly, about the same
diameter as in the first-named situation.   At the apices of the roots
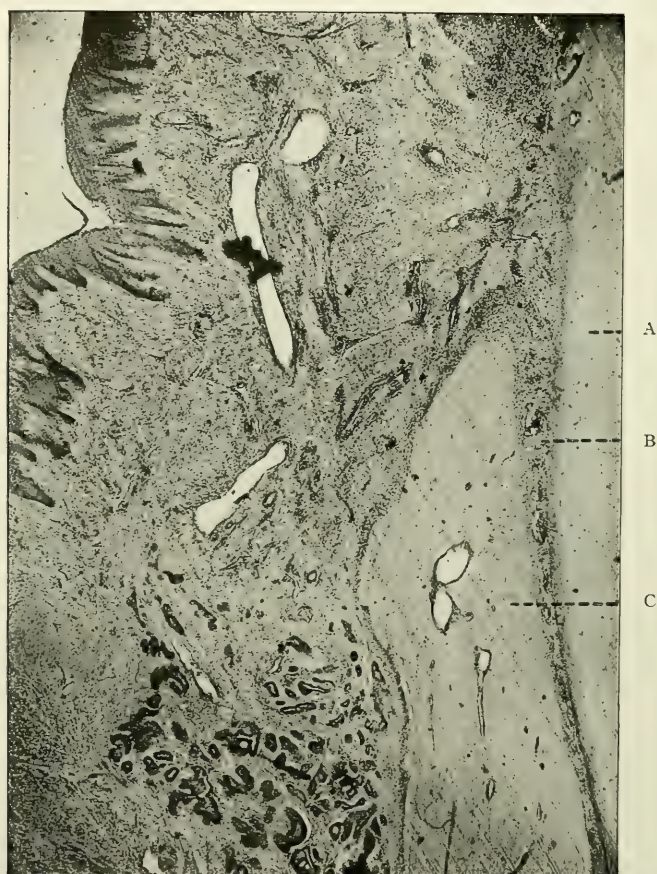
FIG. 267.—Normal bone, internal alveolar process, from jaw of woman of
twenty-five. A. Dentine; B. Periodontal membrane; C. Normal bone.   Magni-
fied 80 times.

the bony socket is closely approximated to the teeth themselves,
the periodontal membrane undergoing only very slight enlargement,
its width remaining practically the same throughout its whole
extent.   The fibres of the gum around the necks of the teeth are not

as densely arranged as one would expect from the statements of text-books. In no sense do they form a firm annular ligament, as described by Stöhr, binding the soft tissues down to the hard parts. Indeed, a V-shaped space probably always exists to a slight degree in normal circumstances. All human teeth possess spaces—the
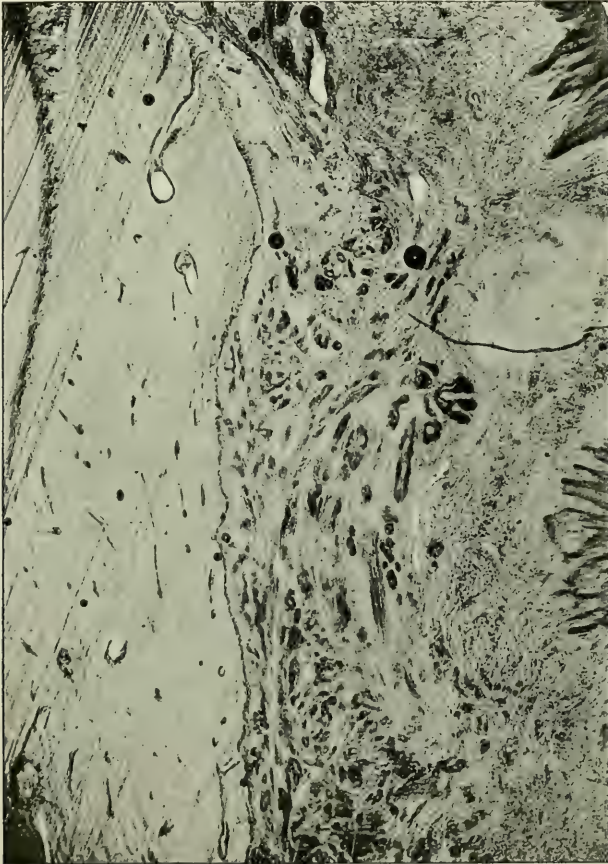
FIG. 268.—Same as preceding illustration, but at cervical region. Normal. Magnified 80 times.

gingival troughs—around their necks, which may become potential cavities for the retention of micro-organisms (see Chapter X, Vol. I).

If great care be exercised in the laboratory manipulations, it will be found that micro-organisms can always be demonstrated, in ordinary circumstances, occupying the site thus produced. It is

when they are of the pus-producing varieties that "pyorrhœa alveo-laris" is established.

EARLY UNSUSPECTED CHANGES PRODUCING OSSEOUS ATROPHY

In the mouths of people of middle age a startling fact may be frequently observed. Without any signs of gingivitis whatever, or the presence of tartar or "pyorrhœa," when the gum tissues are what one would call normal and healthy and the adjacent teeth fully functional and free from caries, decalcification or halisteresis of the free edge of the osseous socket may be beginning (see photo-micrograph, Fig. 269). It must be remembered that the bone forming the dental sockets is peripheral, and—as is pointed out by the author in Vol. I—is structurally different from compact bone, properly formed, elsewhere. This ill-constructed attachment and foundation of the teeth easily and early falls a prey to disturbances in the vascular system of the jaws, and soon begins to degenerate and atrophy, after an acute attack of anæmia, long-continued fevers, rheumatism, hydrargyrism, etc.

Investigated by the author was an affection of the mandibular teeth of a girl of ten years, who suffered from osseous atrophy associated for a few months with "pyorrhœa," which resulted in a permanent loosening of the two first incisors. The rest of the mouth was healthy. There occurred the loss of the bone of the sockets, and the presence of extremely deep pockets around each tooth. The author attributes the affection in this case directly and entirely to an acute attack of anæmia, following chickenpox at three-and-a-half years, which, in addition to producing general symptoms, acted locally by modifying the usual amount and character of the blood supply to the parts and causing malnutrition and degeneration or atrophy. The limitation of the condition to the two teeth named was probably owing to an undue amount of function, the posterior teeth not having erupted. Accentuated use is as bad for the teeth as is idleness. "Pyorrhœa alveolaris" may be confined to one or two teeth, when it will generally be found that they are either used too much—as, for instance, in holding a pipe between them —or to loss of function, as in the case of absence of an opponent in the other jaw.

Sections of jaws with the teeth *in situ* which would be considered as normal, very frequently exhibit this atrophy of the bone. The result is a deepening and a widening of the gingival trough and its
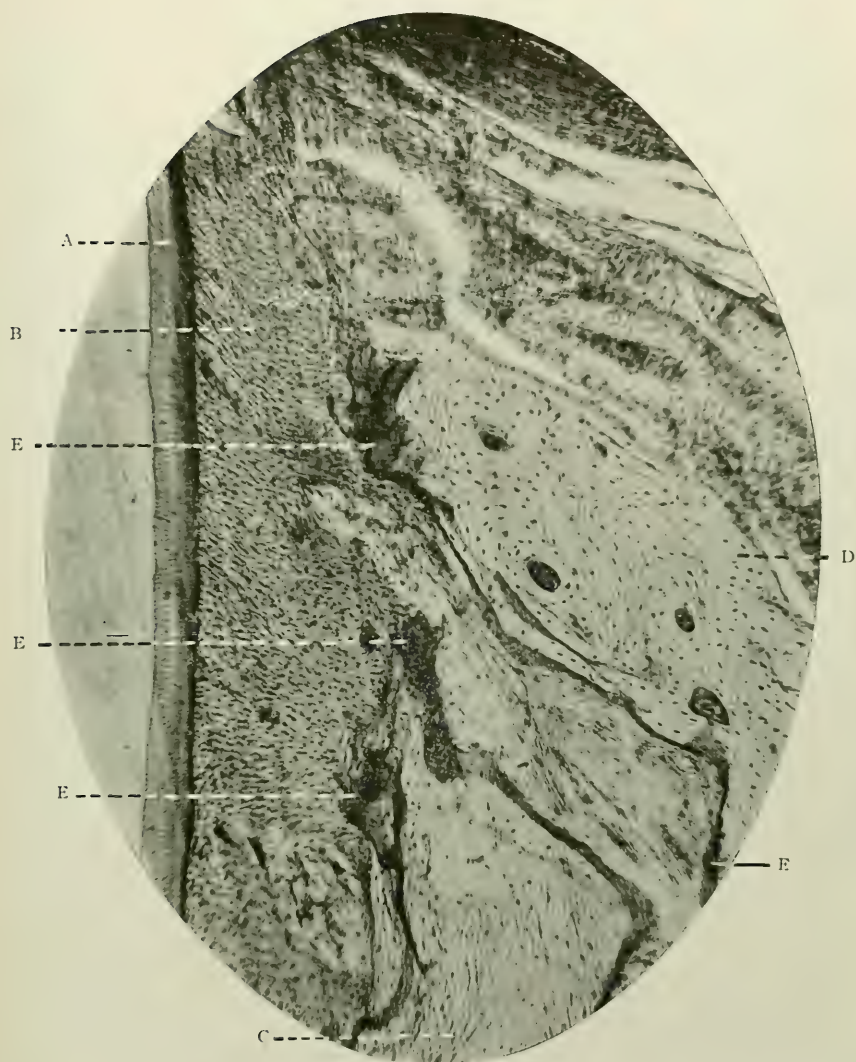
Fig. 269.—Vertical section through cervical region of molar of man, which presented no symptoms of "pyorrhœa alveolaris." A, Cementum; B. Normal periodontal membrane; C. "Gitter-figuren;" D. Normal bone; E. Areas of decalcification. Magnified 350 times.

transformation into a pocket, in which, if pyogenic bacteria happen
to collect and develop, "pyorrhœa alveolaris" is bound to ensue.

FIG. 270.—Mandibular molar showing absorption of one of its roots by means
of granulation tissue developed as a consequence of chronic inflammation of the
periodontal membrane.   (See Fig. 271.)

ABSORPTION BY GRANULATION TISSUE

In order to further test the statement that granulation tissue—
a part of *osteitis rarefaciens*—is not present in the earlier conditions,
a lower permanent molar, with one root absorbed as a result of the
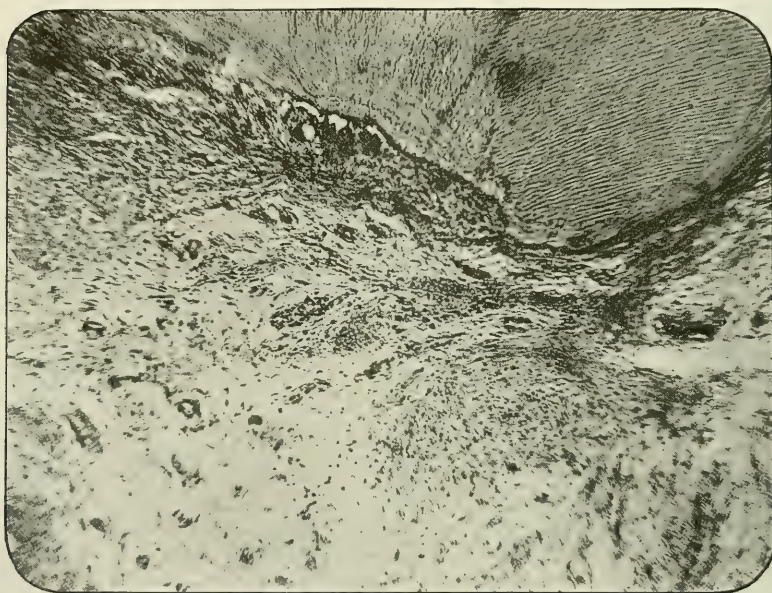
FIG. 271.—Granulation tissue absorbing dentine.

action of certain cells in the granulation tissue produced by an in-
flammation of the periodontal membrane, was examined, with the
microscopical appearances revealed in Fig. 271.

It is important to recognise the fact that granulation tissue

such as occurs in the healing of a suppurating wound, consists of a dense cellular new growth freely supplied with blood from capillary vessels of new formation. The cellular elements comprise: (*a*) Formative cells derived from pre-existing cells of varying shape and size; they may be oval, branched, or oat-like; their nuclei round or oval. (*b*) Polymorphonuclear leucocytes, which take no part in the formation of the new tissue. (*c*) Small lymphocytes and plasma cells with large, round, deeply staining nuclei. (*d*) Large mononuclear hyaline leucocytes, which, according to Metchnikoff, have the prop-

Fig. 272.—For comparison. Radicular portion of firm canine, in maxilla of man aged forty-five. A. Hyperplasic cementum; B. Periodontal membrane. Magnified 40 times.

erty of becoming metamorphosed into connective tissue cells. (*e*) Multinuclear giant cells. The latter occur in granulation tissue of bacterial origin (like tubercle), and in "wounds, around necrotic material, and about foreign bodies." In the photomicrograph the latter are absent. Comparison of this figure with Figs. 263 and 264 shows the difference between the two, and demonstrates the fact that no granulation tissue is present in the sockets of teeth in the earlier stages of "pyorrhœa alveolaris," and does not necessarily always exist even in later stages.

COMPARISON BETWEEN CHRONIC PERIOSTITIS AND SENILE CHANGES

Sections were cut, for further comparison, of the jaw of a male aged forty-five, in which the maxillary left permanent canine was firm, and its distal neighbour, the left first premolar, was extremely loose. This gave an opportunity for observing the differences in the microscopical appearances between the two sockets, the former exhibiting the usual degenerative changes of the periodontal membrane which are incidental to middle age, and the other the abnormal
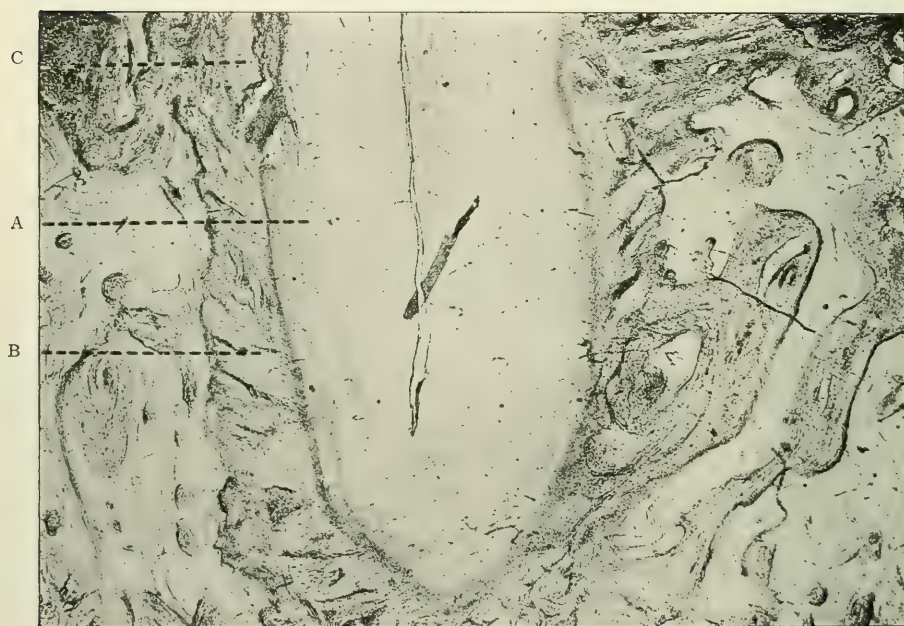
FIG. 273.—For comparison with preceding illustration. A. Hyperplasic cementum; B. Chronic inflammation of periodontal membrane; C. Fracture of cementum mentioned in text. Magnified 40 times.

changes due to chronic periostitis (periodontitis). In the former there are no signs of inflammation; the connective tissue is increased in amount, and is more pronounced than in younger membranes; in the latter, all the histological signs of chronic inflammation are seen. But there is a marked variation from those of "pyorrhœa alveolaris." While the membrane is extremely broad, the myeloid cells and osteoporous spaces of the bone are wanting   Incidentally

these sections also show the fracture of an extremely minute portion of the cementum of the premolar.

<center>"POCKETS"</center>

In Fig. 262 it will be noticed that the free edge of the gingival tissue, as represented by the oral epithelium, is loosely adherent to the terminal margin of the cementum; this is exhibited under a higher magnification in Fig. 274. The enamel has been lost, owing to decalcification in weak *aqua regia*. No hard-and-fast rule exists as to the cervical attachment of the gum tissues from an anatomical point of view. Even as the normal relationships of enamel, dentine, and cementum vary, so do the attachments of the soft parts. The "pocket" in this section is extremely slight; nevertheless, it had contained, during the life of the patient, a mass of micro-organisms, which had induced a slight suppurative gingivitis (E in Fig. 262), and "pyorrhœa" was actually present. At some considerable distance nearer the radicular region of the tooth, the usual transformation of the periphery of the alveolar process of the jaw had already taken place, the bone being converted into osteoid tissue through halisteresis, although the patient was only thirty-nine years of age. This osseous surface was extensively decalcified by a similar process, and the blood-vessels in the periodontal membrane, exercising a protective influence, were hyperæmic. When this is compared with Fig. 265, in which case "pyorrhœa" was absent, it is found that in the latter a deep and extensive pocket had been produced, the gum margin being attached to the cementum at a distance of 1.5 mm. below its free edge (see Figs. 275 and 276). It is not surprising, therefore, that a small-celled infiltration—which is a normal condition of the gum—has occurred in the immediate neighbourhood of the deep socket. But it is an astonishing fact that there is less pathological decalcification of the bone than in the preceding instance, clearly demonstrating that the amount of loss of the marginal bone does not depend upon the depth or shallowness of the pocket. It is important to recall that here no "pyorrhœa" whatever was manifest.

It is unnecessary to lay before the reader, at present, any further histological details, but in support of his views, the author desires to add a note regarding the anatomical and clinical aspects of the subject.
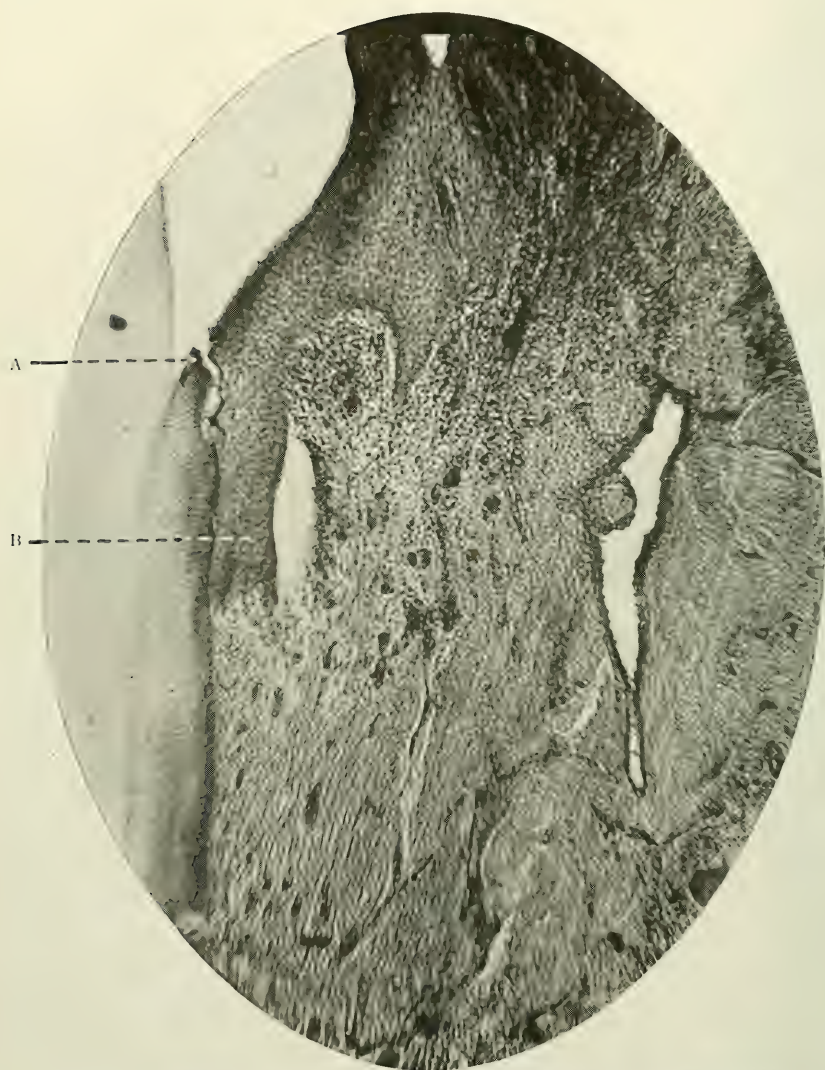
FIG. 274.—Same as Fig. 262. A. Free edge of cementum; B. Oral epithelium of gum tissue. Above A is a shallow pocket filled with pus during life. Magnified 350 times.
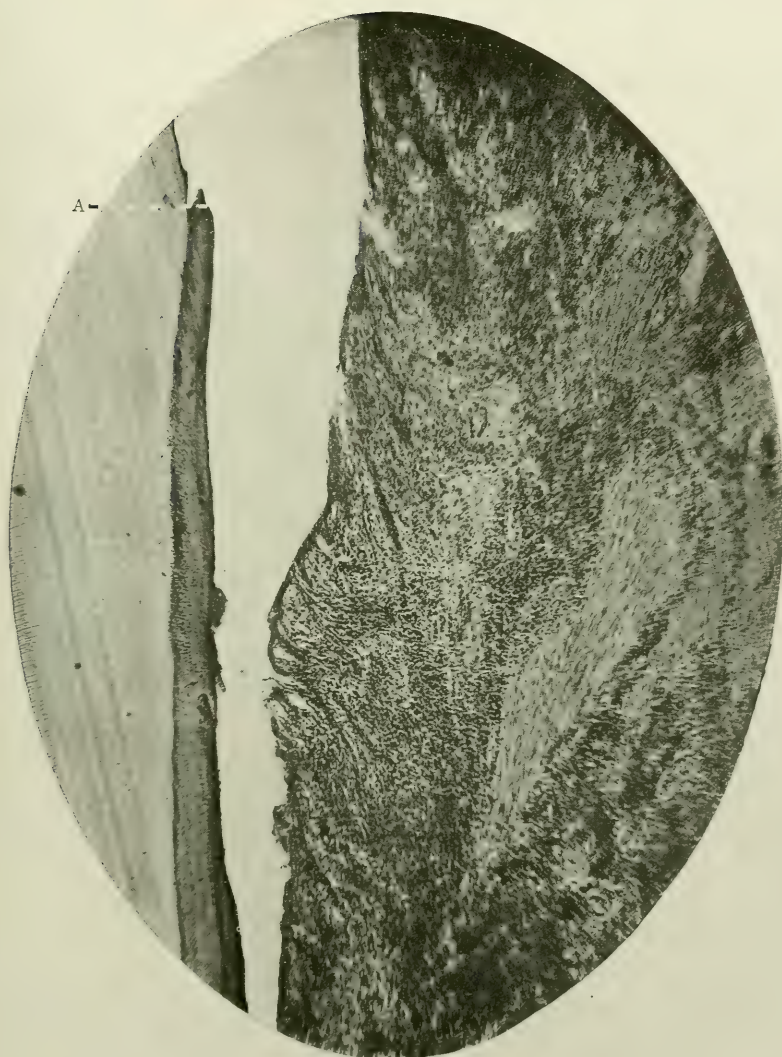
FIG. 275.—Same as Fig. 265. No pus present. A. Free edge of cementum. Gum tissue adherent below. Magnified 350 times. (See next figure.)
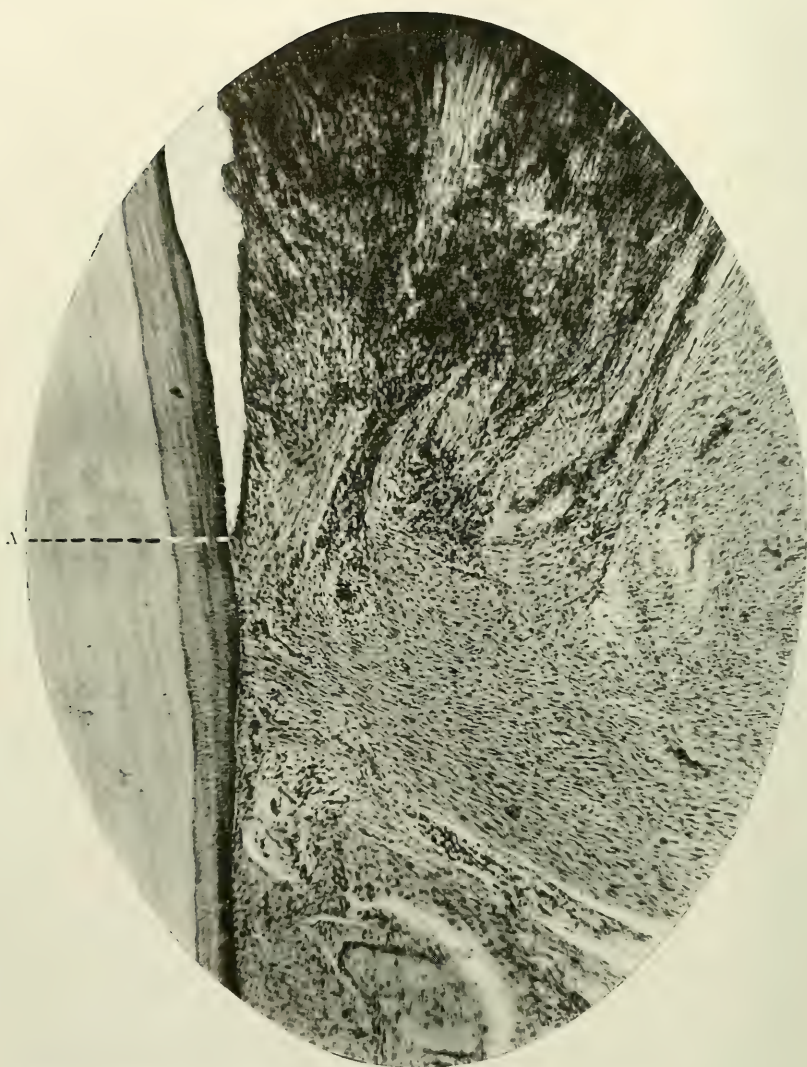
FIG. 276.—Same as preceding illustration. Gum tissue attached to cementum
at A.   Magnified 350 times.

ANATOMICAL AND CLINICAL OBSERVATIONS

Absorption of each alveolar process of the jaws is a common occurrence; it is the rule in man and the lower animals.[1] The more aged the individual the greater the loss of this bone, with the concomitant shedding of its dental occupants. The teeth of dogs, cats, monkeys, and other animals, either in a domesticated environment or *in naturâ fera,* become loose as time passes by, as a direct consequence of the absorption of their sockets—a physiological process. Man becomes more and more inclined to be edentulous as he advances in life, a part of the decadence of his vital powers. Thousands of skulls of aged people exist where the teeth remain *in situ.* This is due to the splendid natural physique of the owners. But in probably eighty per cent. of individuals living in highly civilised communities, it is the normal condition for the alveolar processes to atrophy and shrink, for the gum tissues to become thinner and degenerate, and for the roots of the teeth to become exposed. Many cases of so-called "periodontal disease" are not the outcome of pathological disturbances at all, hence it follows that these senile changes, which may begin early or late in life, may induce, in the first instance, the deepening and widening of the normal trough at the gum margin, and then, if this is infected by pyogenic bacteria, "pyorrhœa alveolaris" follows. Clinically, "pyorrhœa" is unaccompanied by pain, *i.e.,* generally speaking; if complications exist, pain may be present. As a rule, its evidences and effects are unknown to the patient, and its diagnosis, at times, is difficult for the dental surgeon. If it were produced by an *osteitis rarefaciens,* as is so generally believed, it is more than likely that all the signs and symptoms, objective as well as subjective, of chronic periostitis would supervene, and the patient would be duly warned. But this is not so.

From his personal experience of cases in which "pyorrhœa alveolaris" was a prominent symptom, the author is led to the conclusion that the morbid conditions of the jaws which produce the flowing of pus are not the etiological factors of severe metabolic disturbances of the alimentary tract or the vascular system, but that they are part and parcel of them. A strong infection of the oral cavity by means of pathogenic micro-organisms may induce both an extensive "pyorrhœa" in the pockets already deep enough to receive them, in all parts of the mouth, and contemporaneously

[1] See Appendix, Vol. I.

a secondary toxæmia or other lesion which reacts universally on
the bodily tissues. "Pyorrhœa alveolaris" does not initiate but is
produced by the same septic cause which leads to general systemic
affections, and which may set up among other diseases alimentary
toxæmia, gastritis, enteritis, chronic toxæmia, chronic rheumatism
and—remotely—septic anæmia, pernicious anæmia, arthritis, endo-
carditis, septicemia, etc.

SUMMARY

To sum up. The course of events, in the opinion of the author,
is as follows: Atrophy of the bony socket and shrinkage is followed
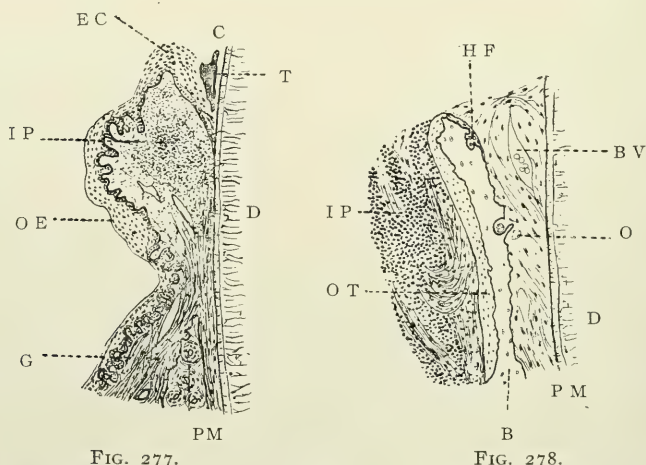
FIG. 277.   FIG. 278.

FIG. 277.—The gum and surrounding structures in "pyorrhœa alveolaris."
Magnified 80 times.   D. Dentine; C. Cementum; P.M. Root membrane; G. Normal
tissue of the gum; B. Normal bone; O.E. Oral epithelium; E.C. Dead epithelium
cells being cast off the ulcerated surface of gum; T. Tartar at gingival margin;
I.P. Inflammatory cells and products. (*After Znamensky, from the Journal of
the British Dental Association.*)

FIG. 278.—Same as the preceding, and from the same source. Magnified
180 times. Lettering as before, but also—O.T. Outer part of socket being trans-
formed into osteoid tissue; H.F. Howship's foveolæ on inside portion; O. Osteoclast;
B.V. Capillary in normal root membrane.

by a widening of the gingival margin and broadening and deepening
of the troughs, with hyperplasia of the periodontal membrane. If
a pathogenic infection occurs, there is a lodgment of pyogenic
bacteria in these already suitable pockets, and "pyorrhœa" results,
and it may or may not be accompanied by gingivitis and the produc-
tion of tartar.

ZNAMENSKY'S RESEARCHES

Znamensky[1] sums up his opinions as follows:—

1. The process in the bone is *osteitis rarefaciens*.

2. The disease develops not *primarily* in the bone, but begins with a suppurative inflammation of the gums, which, approaching the bone, gradually produces therein the above-mentioned condition.

3. An osteoporous form of atrophy of the sockets affords a very favourable nidus for its development.

4. Exhausting diseases create in the alveolar sockets a favourable nidus for the development therein of "pyorrhœa alveolaris" in the form of an osteoporous atrophy.

5. The lack of real hygienic care in regard to the teeth at the time of the foregoing constitutional diseases, and a deposition of tartar give rise to a suppurative inflammation of the gums, which rapidly passes into "pyorrhœa" on the suitably prepared nidus of the bone of the sockets.

6. In slight cases, affecting only that part of the socket which does not contain bone-marrow, the hygienic care of the teeth—such as removal of tartar, washing with disinfectant and astringent lotions, and correct regimen of diet will alone be sufficient to suppress the ailment. In severe cases more stringent and radical measures must be adopted.

HISTOLOGY (ZNAMENSKY)

First steps. Irritation of the gums occurs through depositions of tartar, occupying the normal gingival margins. A very hard swollen rim of gum appears strongly infiltrated with leucocytes. It includes the papillary layer of the gum, the alveolar socket being at first unaffected. Soon the gum loses its superficial epithelium, and an ulcerated surface results. Emigration of leucocytes from the vessels takes place (see Fig. 277).

On reaching the thin edge of bone (which in this situation does not possess any large medullary spaces, and therefore no marrow) inflammatory changes at once begin. On its free rim, which here attains only the thickness of a sheet of note paper, the socket loses lime salts, and undergoes transformation into an osteoid tissue, and afterwards into a fibrous intervening (uniting) tissue. This

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[1] "Alveolar Pyorrhœa—its Pathological Anatomy, and its Radical Treatment." *Journal Brit. Dent. Assoc.*, October, 1902. Also *Trans. Int. Med. Congress*, 1913.

loss of lime salts is shown by the homogeneous character of the bone, absence of lamellæ, and loss of the characteristic outlines of the bone lacunæ and canaliculi. This part is separated by a line of demarcation from the healthy part. This portion of the bony socket is thus transformed into a fibrous tissue, which passes more deeply into an osteoid tissue, which ultimately absorbs the thin bony plate of the alveolar septum.

At this stage the periodontal membrane begins to be affected. Its blood-vessels are much dilated, and leucocytes immigrate in all directions.

Destruction and removal of the thinnest portion of the alveolar plate are brought about thus: Under the influence of the external inflammation of the gum the bone becomes decalcified, then transformed into an osteoid tissue, and afterwards into a fibrous tissue, which is soon thoroughly infiltrated with leucocytes.
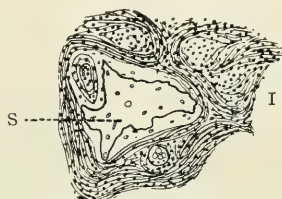
FIG. 279.—The final stage in the development of "pyorrhœa alveolaris." From the same source. Magnified 360 times. The laminæ of the bony socket have been absorbed and transformed into "fibrous intervening tissue," infiltrated with inflammatory cells and products. s. Sequestrum undergoing peripheral absorption; I. Inflammatory infiltration of the tissues.

In those parts of the bone which contain cancellous spaces filled with marrow—i.e., lower down the sides of the cementum of the teeth—the changes just described occur, and in addition a lacunar absorption follows.

This proceeds from the side of the periosteum of the socket, and also from the Haversian systems by means of osteoclasts in the usual way. The foveolæ of Howship are thus produced. Small osseous sequestra are formed, as in Fig. 279.

Thus, Znamensky considers that it is not produced by an atrophy of the sockets of the teeth. Dr. Nikiforoff, quoted by this author, says in his "Pathological Anatomy:" "An atrophical state of the bones shows itself by a thinning or disappearance of bone tissue. In some cases the thinning proceeds as a result of the Haversian canals having become widened, hence termed 'osteoporous.'"

There are two kinds of atrophy of the alveolus originating in the bony tissue itself:—

(1) *Simple atrophy*, in which the socket disappears completely; the roots become denuded; there is no inflammation, and the gum is attached but lightly to the periosteum of the socket; and

(2) *Osteoporous atrophy*, where the bone is thinned at the expense of the Haversian canals, which become widened ("osteoporous"); the gum comes up to the necks of the teeth; there is no denudation of the root. But the teeth begin to loosen on account of the osteoporosis. There is no pus, and the gum is normal.

To conclude. If, however, chronic inflammation of the gum, produced by the presence of tartar as well as some wasting disease, occurs when osteoporous atrophy is already progressing, then alveolar "pyorrhœa" inevitably ensues. (Znamensky.)

CHAPTER XII

DEGENERATION OF THE PERIODONTAL MEMBRANE

MICROSCOPICAL ELEMENTS IN:—(i) The fibrous and cellular tissue; (ii) The areolar spaces; (iii) Changes in the neighbourhood.

INTRODUCTORY

It would seem, *prima facie*, to be a matter of surprise that the periodontal membrane—that thin, structurally insignificant and uninteresting delicate periosteum that covers the roots of the teeth of man—should ever present many or even any pathological changes for examination by the dental surgeon or pathologist, or open up to him new fields for investigation and research, as macroscopically it appears so unimportant and so far beneath his serious contemplation. But second consideration reminds one that it is a fibrous tissue analogous, homologous, and practically identical both anatomically, physiologically, and pathologically with the periosteum of bones. And when this fact is recalled, and thoroughly recognised, it is not difficult to understand that, as a consequence of disturbances in or loss of the functional activity of its cellular and fibrous elements, or as a result of certain metabolic processes occurring therein, it can and sometimes does undergo metamorphoses of disintegration or atrophy on the one hand, or abnormal forms of growth and proliferation on the other; and that incidental to these retrogressive or progressive changes it may, almost as much as the periosteum of the jaws and long bones themselves, inflame, degenerate, or give origin to neoplasms of either a benign or a malignant nature.

The progressive transformations of this tissue have been narrated in Chapter X.

Degeneration of an organ in general pathology usually implies a gradual retrogressive alteration in its component parts, brought about in the first instance in one or more various ways. If cellular protoplasm becomes converted by an abnormal intracellular metabolism indirectly into functionless but non-necrotic and non-irritating tissue, and actual death does not supervene, a form of degeneration of the parts is established. Thus fatty, colloid, mucoid,

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calcareous, or other degenerations are types of well-known changes induced by conversion of the cytoplasm into several kinds of morbid and useless substances, and are often followed by cell-destruction; but the dental pulp, the periodontal membrane, and the soft parts found in the Haversian and medullary canals of neighbouring bones, seem sometimes to be able to undergo degenerative atrophy on which neither necrobiosis nor necrosis can possibly follow.
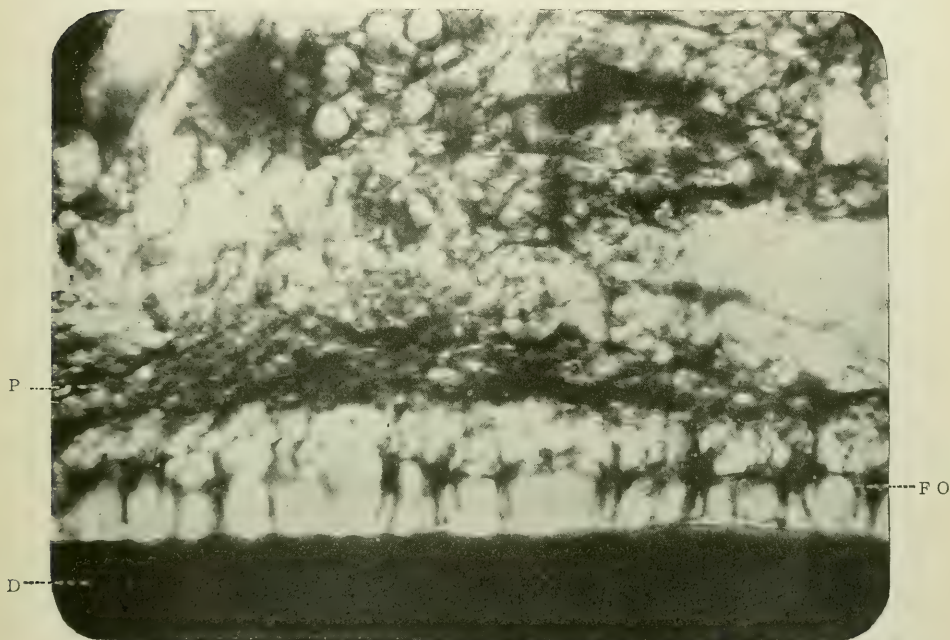
Fig. 280.—Transverse section of fibroid degeneration of the pulp, cut *in situ*. Prepared by "fixing" and hardening in alcohol and formaldehyde, and decalcified by the author's method.   Stained with Ehrlich's acid hæmatoxylene.   D. Dentine with tubules; F.O. Fibroid odontoblasts; P. Atrophied pulp tissue; *Cf.* with following figures.   Magnified 170 diameters.

The disease about to be described is of the nature of a fibrosis or hyperplasia of the individual connective tissue fibres and cells of these soft organs—the precise clinical, histological, or pathological processes which have produced such results being, at present, very obscure and hard to trace.   It is probably due to an exaggerated localised lack of nutrition induced by senile changes in the body generally—not a simple atrophy in which there is a decrease in the size or number of cells or fibres, but a degenerative or marantic or

senile condition where the protoplasm and the nuclei of the cells are altered completely.

Descriptions of fibroid degeneration are not generally found in text-books of pathology, and this probably can be accounted for by the fact that it seems to be limited chiefly to those vascular structures which are situated within or between hard osseous, unyielding walls.

Regarding this from a dental standpoint, these peculiar anatomical relationships obtain only in the mouth and jaws: in the first instance

FIG. 281.—Transverse section of fibroid degeneration of the alveolo-dental periosteum, cut *in situ*. Prepared and stained as in preceding figure. D. Dentine; c. Cementum; A. Alveolar bone; M. Atrophied root-membrane. Magnified 50 diameters.

the pulp inclosed in its dental environment, in the second the root-membrane limited by cementum internally and by alveolar bone externally, and again the medullary tissues surrounded so securely by the concentric lamellæ of the Haversian systems of the alveolar processes of the maxillary and mandibular bones. In this manner the omission may be explained, but that the conditions represented by the term "fibroid degeneration" do exist there is no possible doubt whatever.

The author in 1892 drew attention to a frequent degeneration of
the dental pulp which on comparison with the similar condition of
the periodontal membrane bears a curious, interesting, and instructive
resemblance to the latter.   A casual glance at Figs. 280 and 283 shows
that fibroid degeneration of both are almost homeomorphous.   A
reference to the etiology and patho-histology of the former will
throw some light on the clinical and pathological histories of the
latter.
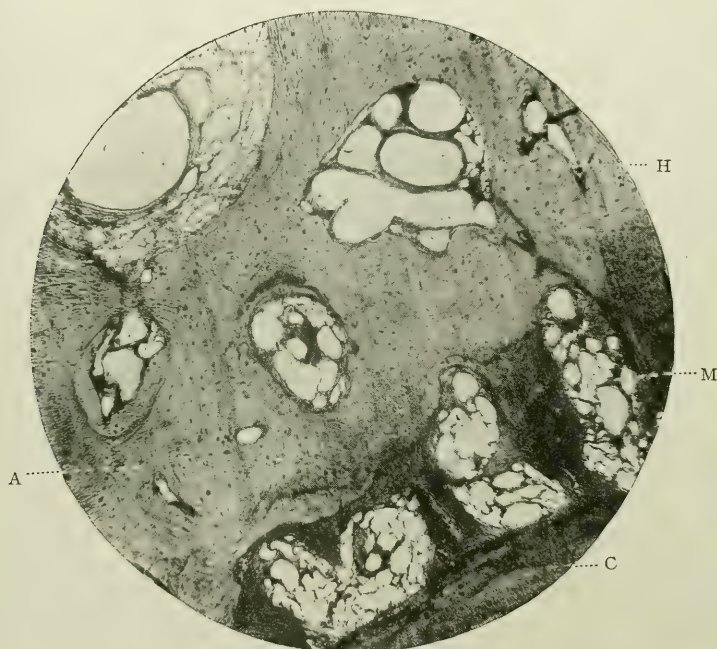
FIG. 282.—Transverse section of the same.   Prepared as in Fig. 126.   Stained
with hæmatoxylene and counterstained with warm ammonia-picro-carmine.
c. Cementum; M. Root-membrane; A. Alveolar bone; H. Enlarged (osteoporous)
medullary spaces.   Magnified 120 diameters.

Briefly, it may be mentioned that fibrosis of the pulp is simply a
"natural old-age termination of the life of a healthy pulp" which
has survived any attacks of an endogenetic or exogenetic character.
It is not dependent, in the least degree, on inflammation of that
organ, but attendant on senile, marantic,[1] constitutional changes.
Several writers in America and on the continent of Europe have
described certain affections which may be allied to or even be pre-

[1] Hektoen and Riesman.   A Text-book of Pathology, vol. i., 1901.
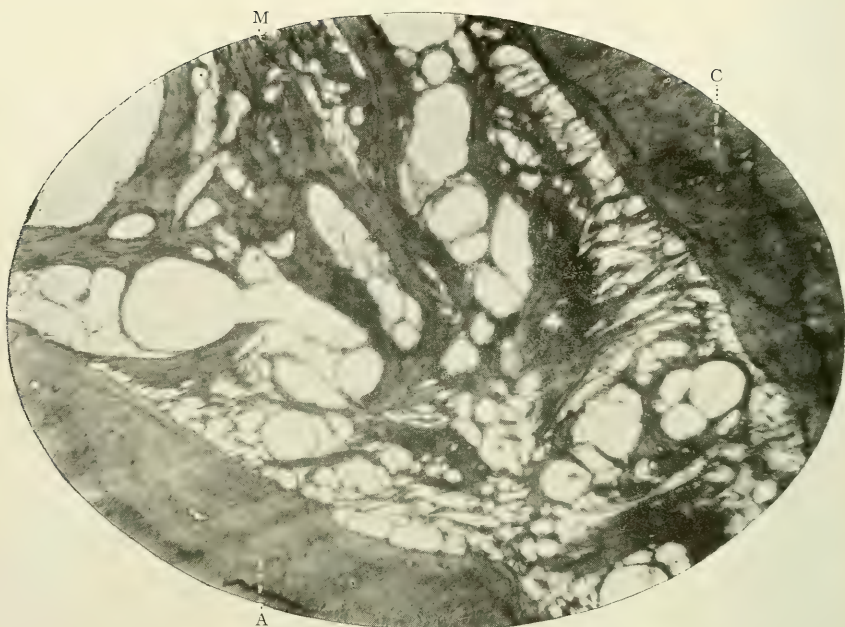
FIG. 283.
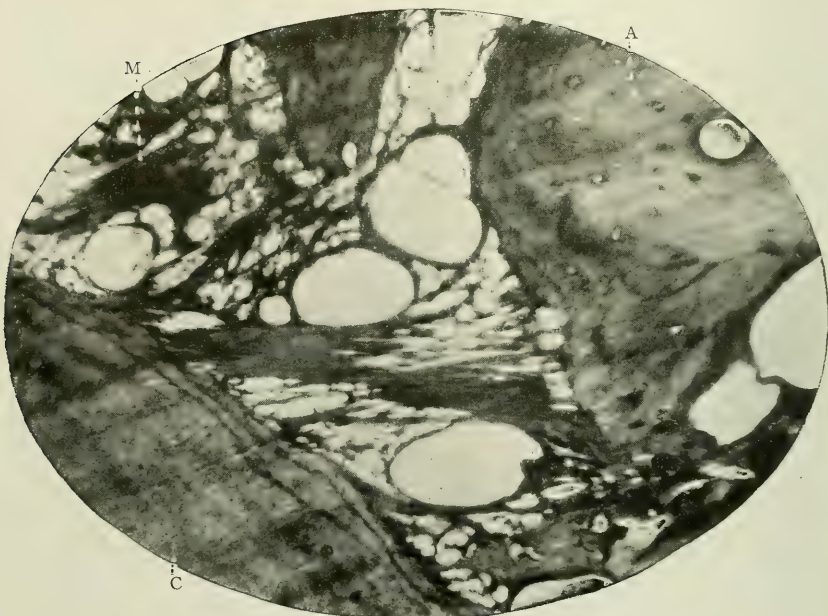
FIG. 284.

FIGS. 283 and 284.—Transverse sections of the same. Prepared similarly. Stained with iron perchloride and tannic acid. c. Cementum; A. Alveolar bone; M. Root-membrane. Magnified 266 diameters.

cursors of this fibrosis, notably the areolation and œdema described by Black, the reticular atrophy of Wedl,[1] and the "*Atrophia pulpæ scleroticans*" of Rothmann.[2]

But in complete fibroid degenerations there are no cells of any description, no nuclei, no odontoblasts, no nerve fasciculi, and no blood-vessels; while the connective tissue, "which is but a loose mass of network in the normal state, has become grossly hypertrophied or quite obliterated, and its place taken by a new, firm, fibrous
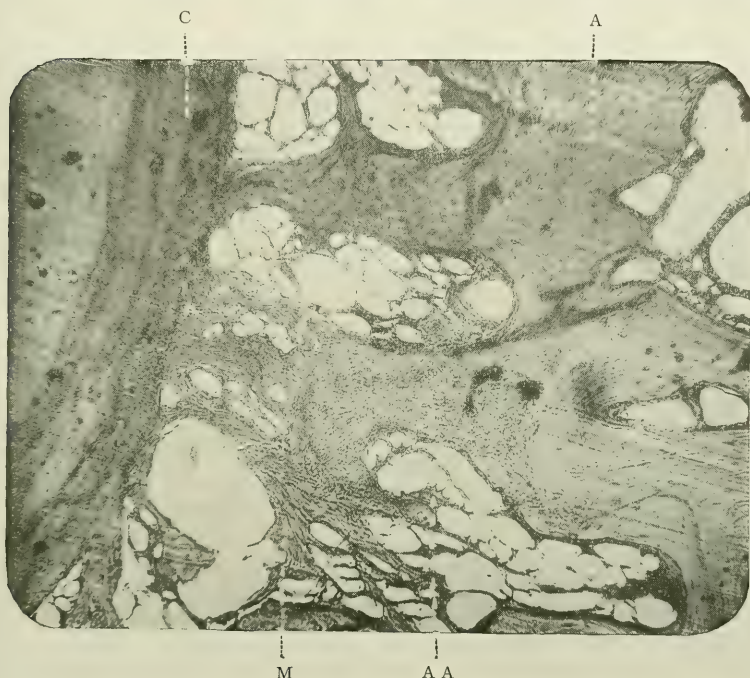
FIG. 285.—Transverse section of the same. Prepared as before. Stained with hæmatoxylin.   c. Cementum; m. Root-membrane; a. Alveolar bone; a.a. Structureless alveolus mentioned in the text.   Magnified 160 diameters.

structure devoid of cells, nuclei, or any regular arrangement of constituent parts:" see Chapter VII, Vol. II.

There is therefore a remarkable coincidence in the microscopical characteristics of complete fibrification of the pulp and the root-membrane; but there are two important differences.   One is that, in the pulp, the whole of the organ becomes simultaneously and

[1] Atlas zur Pathologie der Zahne, 1869.
[2] Pathologie der Zahnpulpa und Wurzelhaut, 1889.

perhaps suddenly metamorphosed, while in the alveolo-dental periosteum, and in a lesser degree in the spaces of the osteoporous alveolar bone, the changes which lead up to a perfect fibrosis can be observed taking place side by side within the same area; the other, that whereas fibrosis of the pulp affects only that tissue, that of the periodontal membrane affects not the cementum, but the bone of the socket, which in its turn shares the innutrition of the vascular periosteum. It may be remarked, however, that fibrosis of the pulp and the root-membrane and the soft tissue in the osteoporous bone may occur synchronously as a result of the general senile changes in the hard parts of the buccal cavity.

An examination of the mouths of elderly people often reveals, associated with absorption of the alveolar processes of the jaws, the presence of sound but aged and yellow teeth, portions of whose roots are exposed. The cementum is laid bare, and the periosteum gone. The teeth may be very loose or fairly firm, free from pain or afflicted with a subacute periostitis as the result of sudden traumatism. Chemical or thermal stimuli yield no effect. If the alveolo-dental membrane of such teeth as these has never been subjected to disease, but has simply run its life-course and has now become atrophied, fibrosis has most likely occurred; and the microscopical study of such a membrane is rewarded by the discovery of certain new histological appearances which can now be described in fuller detail.

HISTOLOGY

(i) *The Fibrous and Cellular Tissues*

In places the fibres are thin and delicate at the edge of the cementum (Fig. 281), but as they unite to pass obliquely outward they increase in diameter and coarseness, here (Fig. 282) being thickly meshed together in broad long bundles or sheaves with but little branching; there, more sparse and sending out inosculating branches (Fig. 283). Their attachment is very strong both externally, and their free extremities seem to be "built into" the hard tissues (Fig. 284). The thinner fibres are structureless, possess clear double contours, and branch freely. A nucleus of a connective tissue cell may, at times, be retained, but the reticular appearance of the thinnest fibres resembles, at a glance, the *stellate reticulum* of the enamel organ minus its cells, at the period when it is about to disappear.

As the thin fibres increase in size, it is seen that they are composed

of fine strands running parallel with one another, until in places they may extend across the intervening space as thick shapeless masses separated from one another by areolæ of varying size and form. (Fig. 285). They pass in a wavy direction similar to the undulating character of white connective tissue fibres, and like them have fusiform, triangular or ovoid cells generally arranged in rows running parallel with the fibres themselves—that is, obliquely outward. Their nuclei are apparently atrophied, and do not ex-
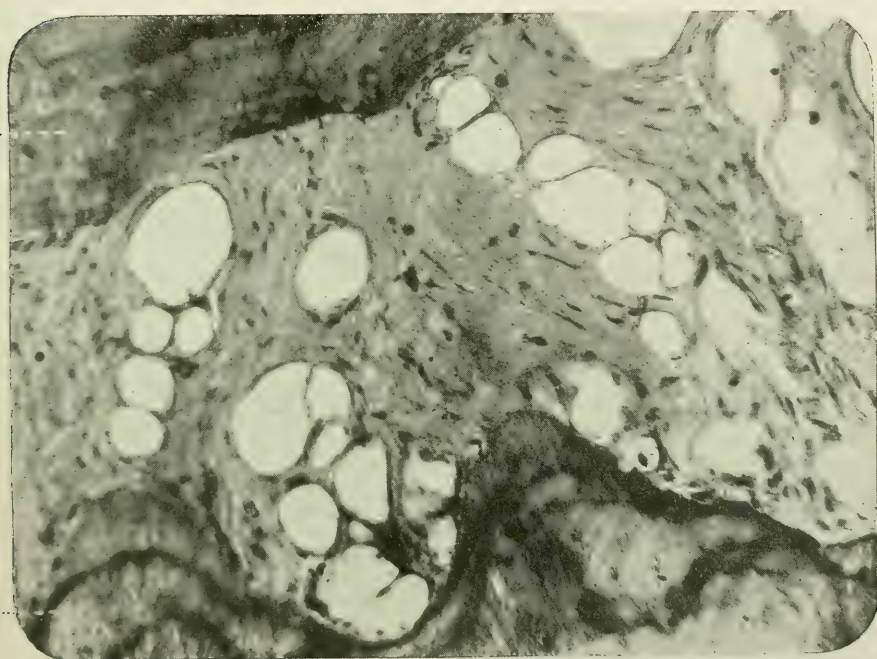
Fig. 286.—Transverse section of the same. Preparation and staining similar. c. Cementum; a. Alveolar bone; f. Fibres with degenerated cells and nuclei. Magnified 300 diameters.

hibit the nucleoli or the karyoplasm or chromatin found in the same cells in a young periodontal membrane—facts well demonstrated when the sections are stained with Ehrlich's acid hæmatoxylene followed by warm ammonia-picro-carmine as in Fig. 282.

These prominent aggregations of connective tissue bundles are probably merely the atrophied remains of the "principal fibres" of Black.

All traces of osteoblasts have vanished absolutely, a few decrepit nuclei alone indicating their anatomical positions in the membrane; and there are no epithelial "rests" of Malassez. There may be a little granular detritus here and there, as also the appearance of fatty degeneration. But this latter most likely marks the commencement of the areolation already mentioned.

(ii) *The Areolar Spaces*

Interesting as are these changes, the most striking point about sections of fibroid degeneration are the areolæ newly developed in the
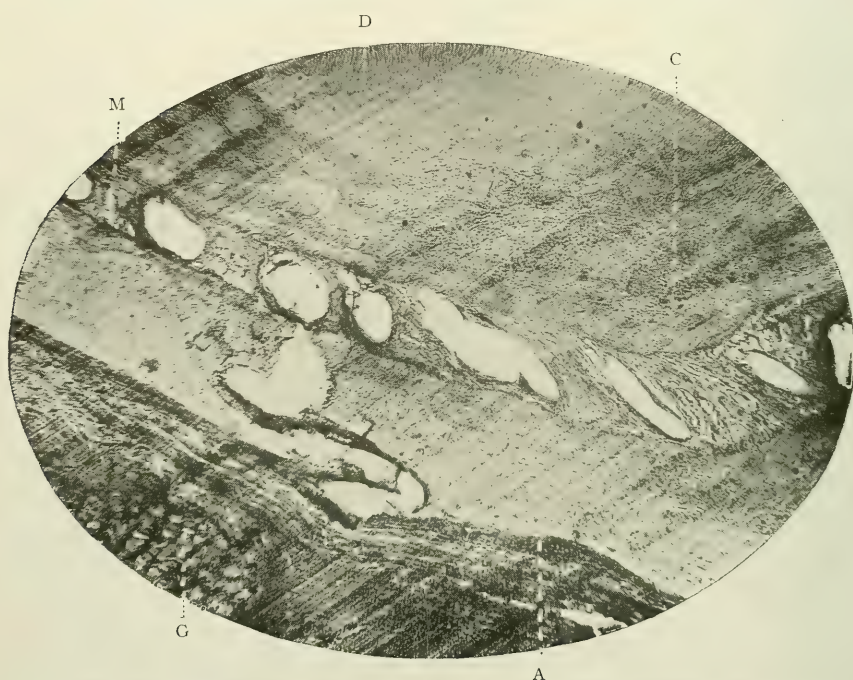
FIG. 287.—Longitudinal section (apical region) of fibroid degeneration of the alveolo-dental periosteum. Preparation, staining, and magnification as in Fig. 285. D. Dentine; C. Cementum; A. Alveolar bone; M. Root-membrane; G. Gum tissue.

tissue. In many instances they extend right across the thickness—or rather the thinness—of the periodontal membrane (see Figs. 281 and 287). The larger ones measure 310μ and more, the smaller 2μ to 20μ, the average being perhaps 60μ.

They are found in great numbers, they vary in shape, being

tubular, oval, or round, and they are bounded and supported by strong curved fibres which pass almost circularly around them.   They are in no sense the remains of the blood-vessels, as they are not confined to the central zone of the membrane, which is, more or less, the rule in normal conditions.   In addition they do not possess the definite walls of arteries, veins, and capillaries.   They are perhaps more marked and obtrusive in transverse than in longitudinal sections (Fig. 286).   Distributed fairly evenly throughout the membrane, they extend into the recesses of the osteoporous alveolus.
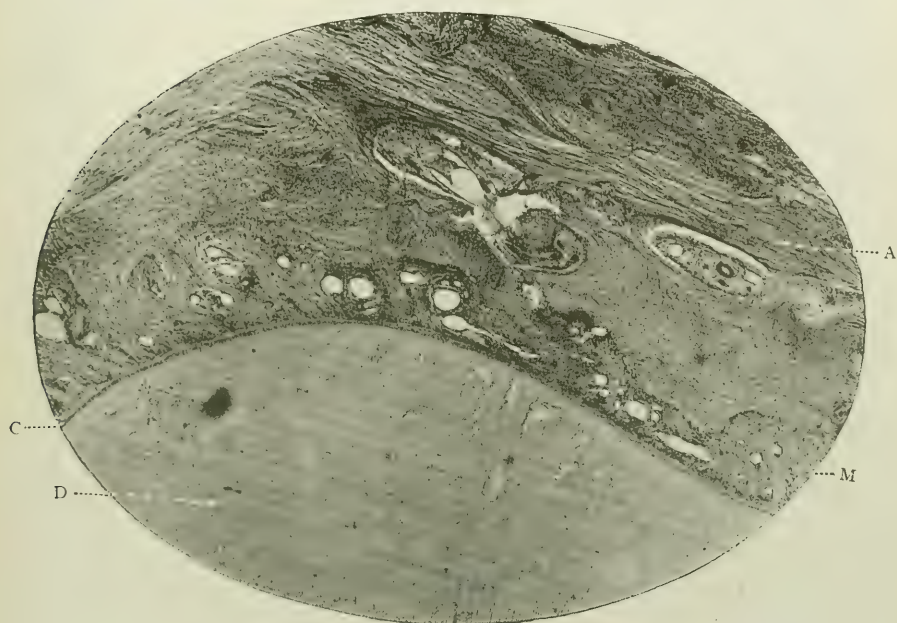
FIG. 288.—Transverse section of the root membrane of an aged tooth. Preparation, staining, and magnification as in Fig. 281, D. Dentine; C. Cementum; M. Root membrane; A. Alveolar bone. *Cf.* cementum in Fig. 281.

The width between the bone and cementum is but little diminished, and differs thus from mere senile changes (Figs. 288 and 289).   It measures in its thinnest portion about 150μ.   But it is a noticeable feature of these sections that the bays or recesses of the alveolar bone are more exaggerated—doubtless not through absorption, as in the case of old teeth affected by "pyorrhœa alveolaris" (see Figs. 290 and 291), but on account of the general osteoporosis which has occurred.

(iii) *Changes in the Neighbourhood*

It is a noteworthy fact that the cementum, although slightly thicker than normal, is not hyperplasic, the majority of the sections cut by the author being free from lacunæ and canaliculi (Figs. 281, 284, 286, and 292).

The osteoporosis of the alveolus is very pronounced, and the enlarged Haversian canals are filled with a shrunken fibroid tissue of a
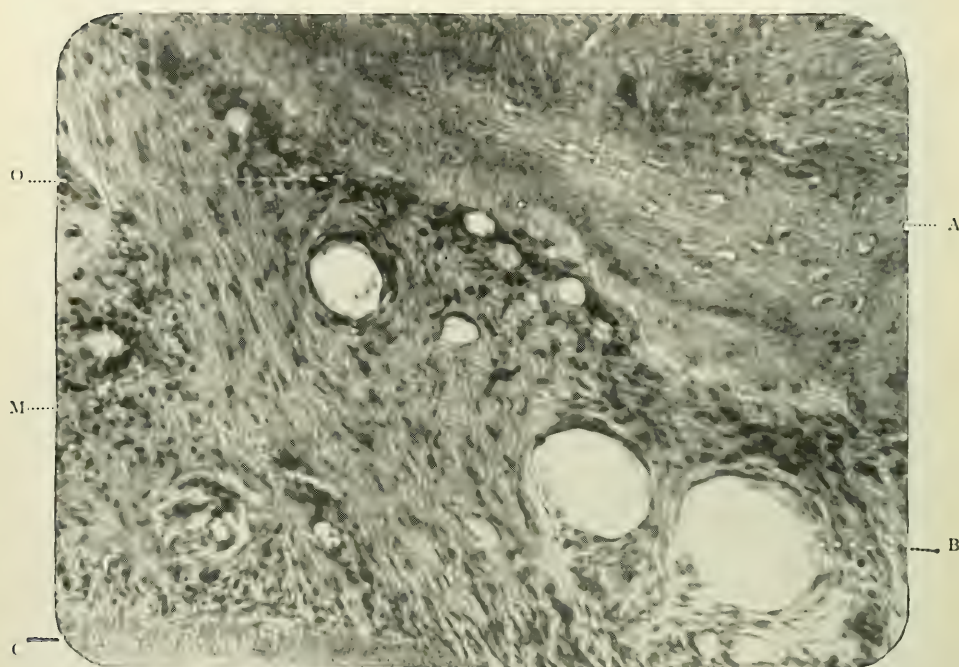
Fig. 289.—Transverse section of periodontal membrane of the tooth with acute "pyorrhœa alveolaris," from the mouth of a man aged seventy-one years. Prepared and stained as above. c. Cementum; a. Alveolar bone; m. Root-membrane; b. Blood-vessels; o. Osteoblasts. Magnified 260 diameters.

character resembling that which is found between the "principal fibres" of the root membrane. Most of these spaces in the bone, which are usually rounded, exhibit one or more large coarse areolæ, possibly and probably the remains of the vascular system (Fig. 293). There are no red marrow cells, no myeloplaxes, no connective tissue cells, no blood corpuscles—nothing but an innutritious non-typical reticulum of coarse and fine fibrous bundles.
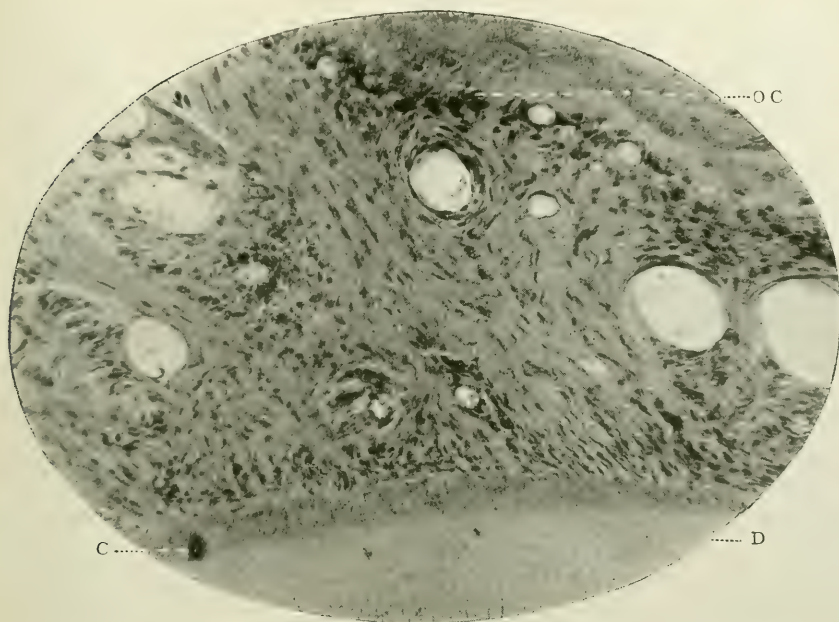
FIG. 290.—Same as preceding. c. Structureless cementum; d. Dentine; oc. Osteoclasts. Magnified 220 diameters.

FIG. 291.—Same, prepared by Weil's balsam method. d. Dentine; c. Structureless cementum; m. Root-membrane; a. Osteoporous alveolus; f. Howship's foveolæ, produced by absorption by means of the osteoclasts. Magnified 120 diameters.
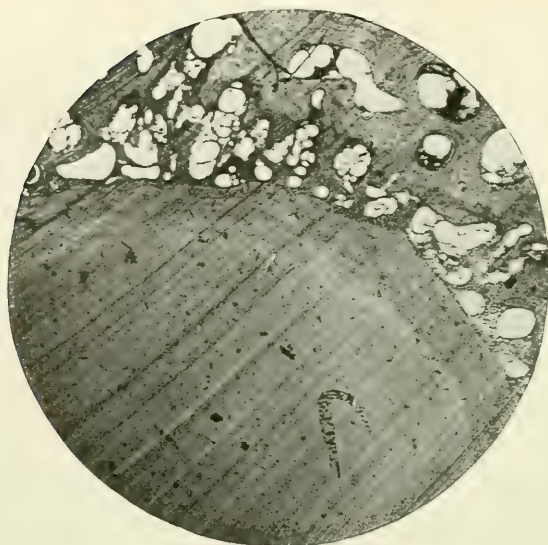
FIG. 292.—Transverse section of fibroid degeneration of the alveolo-dental periosteum. Stained with iron perchloride and tannic acid. Shows the general appearance of the tissue. Magnified 50 times.

FIG. 293.—Osteoporous alveolar bone. Stained with hæmatoxyline. c. Concentric lamellæ; i. Intermediary lamellæ; h. Enlarged Haversian canal or cancellous space; f. Fibroid degeneration of the medullary tissue; b. Atrophy of artery (?) Magnified 250 diameters.

The lacunæ of the Haversian systems are generally speaking abrachiate; and that part of the bone itself which is immediately contiguous to the periodontal membrane has undergone microscopical alteration and degeneration in which the structure of the concentric and intermediary lamellæ are not only masked, but in some instances entirely lost and unrecognisable (see Fig. 285).

There is no calcification of any of the parts of the root membrane, no progressive ossification or osteoplastic signs, no attempts at ankylosis; everything points to retrogressive changes pure and simple.

It would be indeed surprising if, all the other soft tissues degenerating as has been seen, the gum in the immediate vicinity should escape. It does not, but partakes, in its turn, of the general effects of the loss of nutrition (Fig. 287). It is only necessary to add that it becomes much attenuated both in its epithelial and sub-epithelial portions, more coarsely fibrous and less vascularised than usual, and may, wholly or in part, undergo fatty, fibroid, or other old-age changes.

PART II

THE ORAL TISSUES

THE PATHOLOGICAL CONDITIONS OF THE GUMS, PALATE, ANTRUM, AND JAWS

MICROSCOPICAL ELEMENTS IN:—(i) Inflammation of the gum; (ii) Hypertrophy of the gum; (iii) Fibroma; (iv) Spindle-celled sarcoma; (v) Round-celled sarcoma; (vi) Giant-celled sarcoma; (vii) Melanotic sarcoma; (viii) Endothelioma; (ix) Papilloma; (x) Hæm-angioma; (xi) Osteoma; (xii) Adenoma; (xiii) Carcinoma; (xiv) Syphilis; (xv) Inflammation and carcinoma of lining membrane of the antrum of Highmore; (xvi) Tumours of the jaws.

The following diseases of the soft parts of the buccal cavity and its accessory sinus are among the more common affections which come under the immediate notice of dental surgeons. It is not easy to arrange them in anatomical order, and their classification in an alphabetical list is inconvenient. They are here briefly described from the clinical and pathological standpoints, which, it is hoped, will be of service to the reader.

A

OF THE GUMS AND PALATE

(i) *Inflammation of the Gum*

Inflammation of the gum (*gingivitis*) may be acute or chronic, diffuse or local (marginal).

Causes.—Chronic irritation from the presence of foreign bodies such as tartar, edges of fillings, ill-fitting metallic crowns or clasps. "The blue line" of chronic lead poisoning is a chronic general gingivitis.

HISTOLOGY

The soft tissues are infiltrated with inflammatory cells and products. The oral epithelium may be unaffected and the terminal free edge of the alveolar process, at first is unabsorbed. The vessels are hyperæmic.
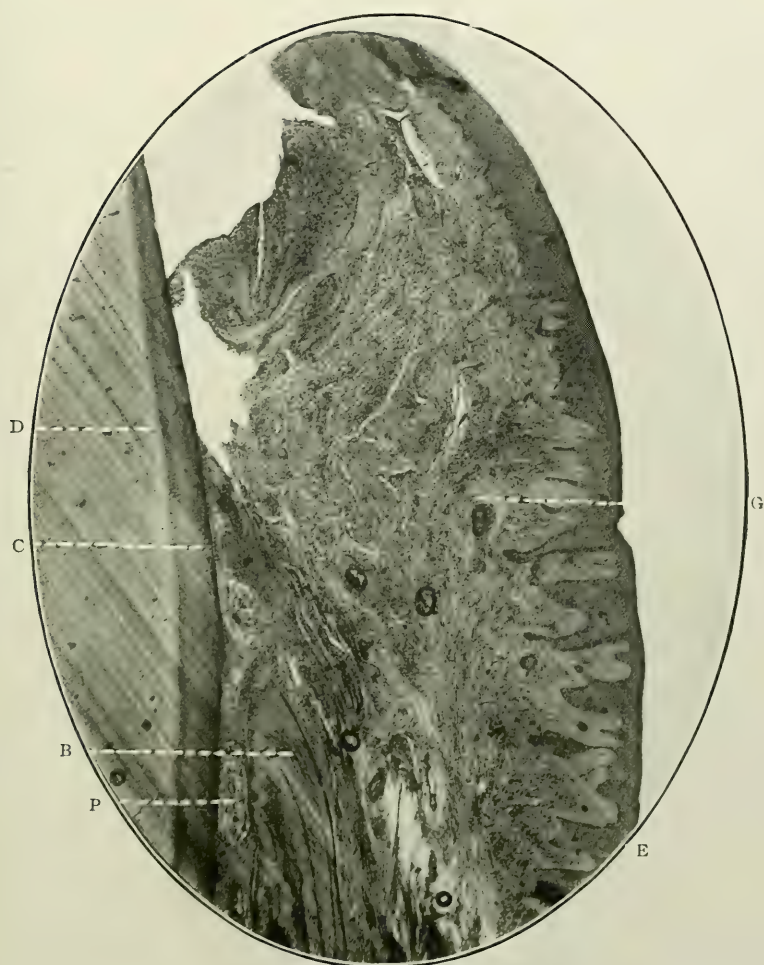
FIG. 294.—Acute gingivitis round a loose second incisor in mouth of a woman aged fifty. Stained with hæmatoxylene and eosine. D. Dentine; C. Cementum; B. Edge of alveolar bone; E. Oral epithelium; G. Inflamed gum; P. Periodontal membrane. Magnified 45 times.

(ii) *Hypertrophy of the Gum*

GENERAL CHARACTERISTICS

Definition.—A non-inflammatory localised increase of the substance of the gum, chiefly apparent round the necks of the teeth,
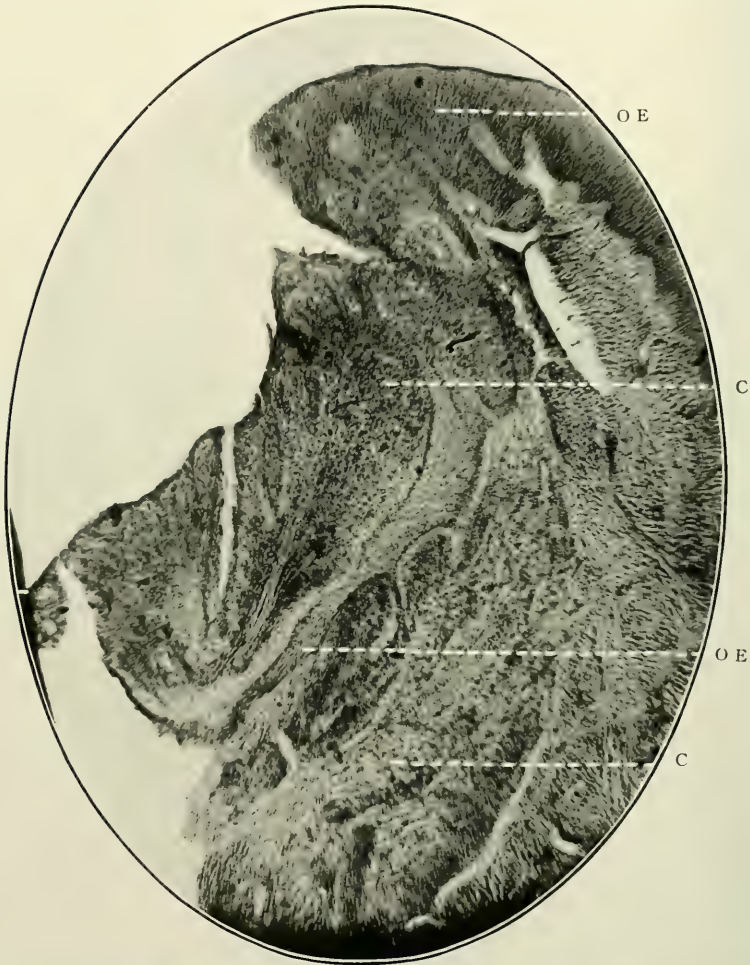
FIG. 295.—The same as the preceding figure. Magnified 200 times. O.E. Oral epithelium; C. Cellular infiltration of the sub-mucous tissue.

due to augmentation of the size or of the number of its cells, or of both, without any appreciable alteration in its structure. *Synonym:* "Polypus" of the gum—an illiterate term.

Its *etiology* is but imperfectly understood.   Some forms, occurring in the mouths of young children, suggest congenital origins; but acquired forms are certainly due to functional increase and nutri-

FIG. 296.—A mandibular molar presenting a true hypertrophy of the gum associated with the periodontal membrane at its cervical margin. A. Shows the new, pedunculated growth, dislodged from the carious cavity into which it had extended—thus simulating a chronic hyperplasic inflammation of the pulp, and its periosteal attachment. B. Shows the position it occupied normally in regard to the tooth itself.

tive supply from any cause whatsoever. Localised hypertrophies are often induced by local irritation, as from tartar, "pyorrhœa
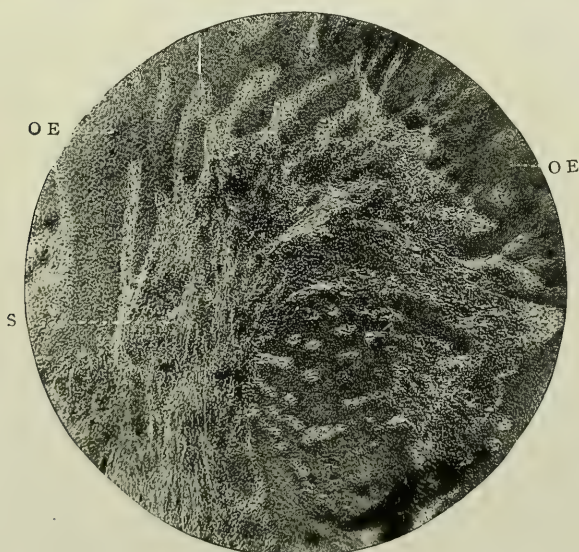
FIG. 297.—Hypertrophy of the gum. Prepared by "fixing" and hardening in formalin and alcohol. Stained with Ehrlich's acid hæmatoxylene. Magnified 45 times. O.E. Oral epithelium; S. Submucous tissue.

alveolaris," etc.   Such cases are not true examples of the condition, but may be termed "inflammatory hypertrophies."

HISTOLOGY

The mucous membrane is generally unaffected, and the sub-mucous tissue may project into it in the form of papillæ, or these may be absent.

The bulk of the tumour is composed of connective tissue fibres, which, somewhat coarser than usual, interlace in every direction. While, in some instances, the vascular system is largely increased in the sub-epithelial regions, the cellular elements, scanty in the nor-mal gum, are much multiplied. Here are found many connective tissue cells, crowds of leucocytes (polymorphonuclear neutrophiles,

FIG. 298.—Similar to the preceding. Preparation, staining and magnification the same. c. Coarse connective-tissue fibres.

eosinophiles and lymphocytes), some mast cells (*Mastzellen*), many of the plasma-cells of Unna, and many lymphoid cells. The leuco-cytes are seen in the tissue spaces, around the blood-vessels, and lie between the coarse fibres of connective tissue. Unna's cells may be scattered throughout the substance of the growth, and the mast cells (whose granules are capable of becoming specifically stained by means of the basic aniline dyes, *e.g.*, gentian aniline violet), can be noticed at the advancing margins of the new growth.

In addition, sections will sometimes show clusters of cells which, at first sight, resemble the epithelial cell-nests which are patho-

gnomonic, when found in certain situations in carcinomata, mixed up with lymphoid cells and a certain amount of fat cells.

Roe, of Philadelphia, examined sections of hypertrophy of the gum from a bacteriological standpoint, and found,[1] on using Weigert's staining method, many *saccharomyces* present in the tissues.

He concludes (p. 350):—"The pathology . . . . . indicates that this disease is not a true hypertrophy, but should be classed with the infectious granulomata; and, in keeping with the established nomenclature, I would propose calling it *Saccharomycosis*, caused by saccharomycetes."

A

TUMOURS OF THE GUMS AND JAWS

Definition.—A tumour is a new growth, not produced by inflammation or mere hypertrophy of pre-existing tissue, which shows no tendency to undergo spontaneous cure or yield to the action of drugs.

A *homologous* tumour resembles and grows in the tissue in which it originates; *e.g.*, fibroma of the periodontal membrane.

A *heterologous* tumour originates in one type of tissue and retaining the features of that type, invades and replaces another type; *e.g.*, carcinoma of the gums, palate, etc.

Clinically, it is either (1) innocent or malignant, and (2) cystic or solid.

Innocent or *benign* tumours, differ from malignant tumours in the following particulars:

DIFFERENTIAL DIAGNOSIS OF

Innocent tumours	Malignant tumours
1. Grow slowly.	1. Grow rapidly.
2. Resemble fully-formed tissues.	2. Do not.
3. Encapsuled.	3. Non-encapsuled; infiltrate neighbouring structures.
4. Movable.	4. Fixed.
5. Lymphatic system not involved.	5. Involved.
6. No dissemination in distant organs.	6. Spread by embolism.
7. No constitutional symptoms.	7. Cancerous cachexia.
8. No recurrence after removal.	8. Often recur.

[1] *The Dental Cosmos*, p. 347, 1901.

Histologically, tumours are classified as

I. *Connective tissue tumours.*

    A. Fully-formed connective tissue type, *e.g.*, fibrous, fatty, cartilaginous, osseous, etc.

    B. Complex connective tissue type, *e.g.*, blood-vessels, lymph vessels, etc.

    C. Young or embryonic tissue type, *e.g.*, round cells, spindle-cells or giant cells.

II. *Epithelial and Glandular tumours.* Squamous, spheroidal, or cylindrical (columnar) type.

III. *Teratoma.* A rare tumour, generally of the ovaries, containing teeth, hair, pultaceous secretions, etc. See Chapter XVII.

They are thus named:

I. A. Fibroma, lipoma, enchondroma, osteoma, papilloma, etc.

    B. Angioma or hæmangioma or nævus, lymph-angioma, lymphadenoma, endothelioma, etc.

    C. Round-celled sarcoma, spindle-celled sarcoma, mixed-celled sarcoma, giant-celled or myeloid sarcoma.

II. Squamous-celled carcinoma or epithelioma. Cylindrical-celled carcinoma.

    Adenoma.

DIFFERENTIAL DIAGNOSIS OF

Sarcoma	Carcinoma
1. Youth and early age.	1. Old age.
2. Each cell is completely surrounded by varying amount of intercellular material, which does not form alveolar spaces; blood-vessels ramify amongst the cells.	2. Cells in alveolar spaces; blood-vessels ramify in connective tissue stroma.
3. Disseminates by blood-vessels* (veins).	3. Disseminates by lymphatic system.
4. Hæmorrhages frequent.	4. Hæmorrhages infrequent.

INFLAMMATORY AND NON-INFLAMMATORY SWELLINGS OF THE JAWS

These may be classified as arising in

A. The *Maxillæ:*

    1. Those involving the *maxillary sinus.*

    2. Those involving the *alveolar process.*

    3. Those involving the *palate.*

* Except in the case of the tonsil, testis, and thyroid body.

B. The *Mandible:*

    1. Those arising from the mucous membrane and periosteum.

    2. Those arising between the external and internal alveolar plates.

<div align="center">A</div>

<div align="center">SWELLINGS OF THE MAXILLÆ</div>

I. Swellings of the antrum are

    1. Those arising locally:

        (*a*) *Fluid*—mucous cyst.

        (*b*) *Solid*—adenoma, sarcoma, carcinoma.

    2. Those arising by invasion: fibroma, enchondroma, osteoma, round, spindle-celled and melanotic sarcoma, carcinoma.

DIFFERENTIAL DIAGNOSIS OF FLUID SWELLINGS OF ALVEOLAR PROCESSES

	Acute abscess	Chronic abscess	Dental cyst	Follicular odontome (simple)	Epithelial odontome
1. Age	Any.	Any.	Adult.	Child.	Young adult.
2. Pain	Great.	Not great.	Slight on pressure.	None.	None.
3. Rate of growth.	Rapid.	Rapid at first; varies.	Slow; progressive	Slow; progressive	Very slow.
4. Appearance of mucous membrane	Acutely inflamed.	Inflamed.	Normal.	Normal.	Normal.
5. Definition	Large area.	Small area.	Small area; globular outline.	Large area; well defined.	Large area; not well defined.
6. Walls	Thin.	Thick.	Very thin.	Very thin.	Thin.
7. Fluctuation	Non-elastic	None.	Elastic.	Elastic.	None.
8. Tooth	Carious and septic.	Carious and septic.	"Dead."	Absent.	Absent.
9. Radiograph	Outline clearly defined.	Outline not so clearly defined.	Very clear outline.	Good definition; tooth in cavity.	Not very good definition; tooth generally absent.

II. Swellings of the alveolar processes are
    1. Those arising from surface—fibroma (so-called " epulis ").
    2. Those arising from interior:
        (a) *Fluid*—acute abscess, chronic abscess, dental cyst, follicular odontome, epithelial odontome.
        (b) *Solid*—fibroma, calcified odontome, sarcoma, carcinoma.
III. Swellings of the palate are
        (a) *Fluid*—acute or chronic abscess from second maxillary incisor, dermoid cyst of soft palate. Rarely, simple follicular odontome, and aneurysm of descending palatine artery.
        (b) *Solid*—innocent tumours, viz., fibroma, papilloma, adenoma, osteoma. Malignant tumours, viz., sarcoma and carcinoma.

DIFFERENTIAL DIAGNOSIS OF SOLID SWELLINGS OF THE PALATE

	Rate of growth	Characteristics	Frequency of occurrence	Situation
Fibroma............	Slow.	Pedunculated.	Common.	
Osteoma (*Torus palatinus*)	Exceedingly slow.	Flat.	Common.	Hard palate.
Adenoma............	Slow.	Sessile.	Rare.	
Sarcoma............	Very rapid.			Hard palate.
Carcinoma..........	Very rapid.			Soft palate.

B

SWELLINGS OF THE MANDIBLE

1. Of mucous membrane or periosteum: fibroma, enchondroma, osteoma, sarcoma and carcinoma.
2. Of interior:
        (a) *Fluid*—acute and chronic abscess, dental cyst, follicular odontome, epithelial odontome.
        (b) *Solid*—sarcoma (usually myeloid), squamous-celled carcinoma.

DIFFERENTIAL DIAGNOSIS OF SWELLINGS OF EXTERIOR

	Situation	Skin or mucous membrane
Fibroma..............	Median line.	Not involved.
Enchondroma..........	Inner side in premolar region.	Not involved.
Osteoma..............	Inner side in premolar region, or at angle.	Not involved.
Sarcoma..............		Ulcerates primarily or secondarily.
Carcinoma............		Ulcerates primarily.

For further signs and symptoms, see above.

DIFFERENTIAL DIAGNOSIS OF SWELLINGS OF INTERIOR

	Rate of growth	Extent	Appearance
Fluid	Slow.	Involves outer plate.	
Solid.........	Rapid.	Involves both plates.	May simulate necrosis of bone.

The salient histological characteristics of the above tumours must now be described.

I. TUMOURS OF THE FULLY-FORMED CONNECTIVE TISSUE TYPE

(iii) *Fibroma*

GENERAL CHARACTERISTICS

Definition.—A connective tissue tumour of the homologous type, arising from the osseous tissues (or their periosteum) of or underneath other parts of the buccal cavity. *Synonym:* "Fibrous epulis."

The term "epulis" (ἐπι upon, ουλα the gums) is often carelessly applied to a true fibromatous type of neoplasm which is of constant occurrence. "Epulis" simply means "upon the gum," and if used at all should have before it a qualifying adjective, such as "fibrous," "sarcomatous," &c. Employed *per se*, it should be deleted from all dental vocabularies.

Etiology.—Congenital influences probably have some share in the formation of these growths; and it is just possible that traumatism may occasionally give rise to them.

With regard to the first hypothesis, it has been suggested[1] that as far as the mandible is concerned they may have their origin in some embryonic "rest," left at the time of fusion of the two halves of Meckel's cartilage; and as far as the palate is concerned, they may take their rise from the sutures existing in the fœtus, between the premaxillary and maxillary bones. This theory thus corroborates J. G. Turner's researches on the subject.

Fig. 299.—Fibroma of the jaws, rising from the interdental osseous septa. Prepared, stained and magnified as in Fig. 297. The "hard" variety.

Situation.—Fibromata are usually found between neighbouring teeth, which may become separated, or springing from the labial or buccal surfaces of the gum covering the alveolar processes of the jaws. Arising from and rarely attached to the periodontal membrane, they may be removed during the extraction of teeth (see Figs. 239, 240 and 241). They are often associated with the periosteum of the interdental septa or that of the jaws.

[1] J. H. Targett, "The Pathology of certain growths about the Lower Jaw." *Trans. Odonto. Soc. of Great Britain*, May, 1902.

Pathology.—In the mouth, as elsewhere, two kinds may be found—the soft and the hard.   They differ in that the former is much more vascular, and has its constituent parts more loosely arranged than the latter.   They agree in the following particulars:—

In shape they are circumscribed masses roughly spherical or oval in outline.   They may be sessile or pedunculated, nodular, or flattened.

Varieties.—Classified pathologically, they are called "periosteal" and "endosteal."   Of firm consistency, on sectionising they show,
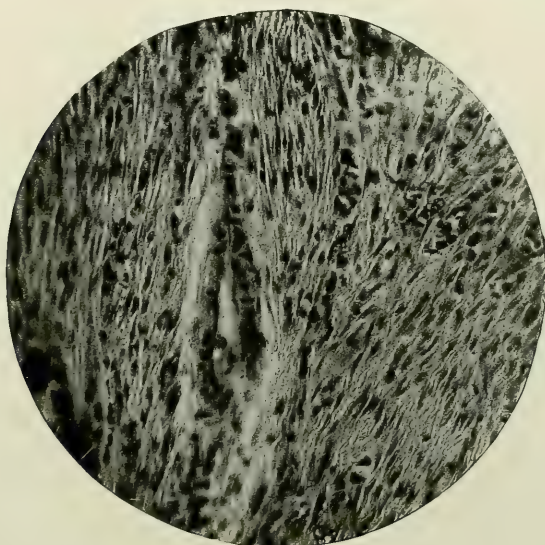
Fig. 300.—The same.   Magnified 250 times.

when examined in the gross, a white glistening surface, with strong, pale bands of connective tissue fibres.

In the mouth they are usually single.

Secondary Changes.—Superficial ulceration, and calcification, wholly or in part.   Rarely hydropic degeneration of the epithelium may occur, and lead ultimately to malignancy.   See Chapter XIV.

HISTOLOGY

Sections of hard fibromata exhibit dense bundles of connective tissue fibres (mature normal fibrous tissues), which interlace in all

directions, but in the sub-epithelial region are arranged concentric-
ally. The blood-vessels are few in number, and lymph-spaces are
scanty. The cells have laterally flattened nuclei, and are practically
spindle-shaped in outline.

The soft varieties have a somewhat embryonic aspect, as that of
newly forming connective tissue. The vessels are large and nu-
merous. The cellular elements, with large round or oval nuclei,
abound in great quantities.

FIG. 301.—The same, showing some of the characteristics of the "soft" variety.
Magnified 45 times.

Normal mucous membrane is present on the surface of both
varieties, but the sub-epithelial papillæ show signs here and there
of variations from the normal type.   "Spiny" cells are constant.

(iv) *Spindle-celled Sarcoma*

This is one of the commonest forms of sarcoma, and may be hard
or soft. The spindle-celled tumours of the gums may be quite be-
nign, or may pursue a malignant course, and have a remarkable
capacity for embolic dissemination.

Definition.—A malignant tumour, the commonest of the sarcomata,
which is composed of spindle-shaped cells varying in size, and con-

taining one or more nuclei. . It is less malignant than the round-celled varieties, at first may be encapsulated but later infiltrate the surrounding tissues.   The cells are embedded in a scanty amount of intercellular material.   The tumours may arise in the periosteum of the jaws or the alveolo-dental periosteum, and at times in the dental capsule.   Rarely they are endosteal in origin.

Varieties.—(1) Small spindle-cells; (2) large spindle-cells.

Secondary Changes.—Fibrification, fatty degeneration, small blood cysts, ossification and ulceration if the gingival tissue is involved.

FIG. 302.—Hard fibroma of the hard palate, showing the coarse nature of the growth.   Magnified 250 times.   *Cf.* Fig. 300.

HISTOLOGY

The cells are much elongated, with long tapering polar processes. A supporting stroma is often indistinguishable, so closely set are the cells.   There may be two kinds of these, small and large, as in the round-celled sarcomata.

In the former the constituent parts are uniform in size and appearance, while the latter display many features of polymorphism, round and oval cells being inextricably mixed with the rest of the tissues.   A photograph of such a tumour, which, in spite of due surgical precautions, constantly recurred locally after removal, is shown in Fig. 303.

The nuclei are usually ovoid, with a small amount of karyoplasm. When the cells are large, however, this nuclear network is abundant and fully developed, and may contain one or more nucleoli. In places, giant cells may be produced by segmentation of the cell nuclei.

The new blood-vessels are merely endothelial tubes surrounding the sarcomatous cells. The pre-existent vessels of the supporting reticulum are probably those arteries, veins, and capillaries which possess well-defined walls.
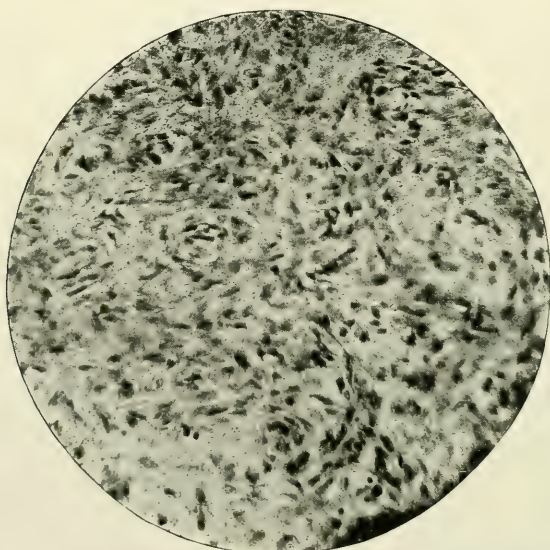
Fig. 303.—Recurrent sarcoma of the jaws.   Magnified 250 times.

(v) *Round-celled Sarcoma*

Definition.—A malignant tumour which resembles in structure most immature connective tissue.

This genus of tumour is of a softer consistency than that just described. It closely approaches the type of embryonic tissue. It is more malignant than the preceding. Originating in the periosteum of the bones of the jaws, it may invade the antrum or attack and absorb the alveolar processes.

In this division two varieties are also found, large and small-celled. In the former polymorphous elements may be discovered: epitheloid and endotheloid cells, arranged in localised masses, and divided from one another by a distinct stroma, may occur, and by

their mode of arrangement give to this form of tumour the name of "Alveolar sarcomata." (Billroth.) These, endothelial in origin, ought really to be classified amongst the endotheliomata.

It is very difficult histologically to differentiate between young granulation tissue and small round-celled sarcomata. The history of the case and the clinical evidence here weigh very heavily in deciding the pathology of the growth and the course of treatment to be adopted, as well as in the general prognosis.

Secondary Changes.—Fatty degeneration or minute hæmorrhages.

(vi) *Giant-celled Sarcoma*

Definition.—A malignant tumour containing giant-cells.

As has been pointed out, myeloid cells may occur at times in the large-celled varieties of spindle and round-celled sarcomata. But giant-celled sarcomata are really those neoplasms not uncommon about the jaws (especially the mandible) of children and young adults, in which giant-cells are a distinguishing feature both with regard to size and number. They are the least malignant of all the sarcomata.

Synonym.—"Malignant 'epulis,'" or myeloma.

Etiology.—These tumours arise probably either from the periosteum of the jaws or the cancellous bone of their interiors. The method of formation of the large masses of protoplasm is obscure. Some of them may possibly be regarded as phagocytes (occasionally blood pigments and other chemical products may be found enclosed in their protoplasm); others possibly have their origin in the fusion or confluence of smaller endothelial cells.

Secondary Changes.—Small numerous hæmorrhages.

It most frequently occurs in connection with bone, is of slow growth, and seldom recurs after removal, in consequence of the fact that it does not invade the lymph nodes nor become disseminated by the blood stream. In the maxilla it arises in the alveolar processes, or the facial wall of the antrum: in the mandible in the alveolar processes, generally (in both jaws) in the region of the second premolar or first permanent molar.

HISTOLOGY

Myeloid sarcoma consists of dense firm masses of round or spindle cells contained in a small amount of fibrous stroma, and possessing large multi-nucleated cells in abundance.

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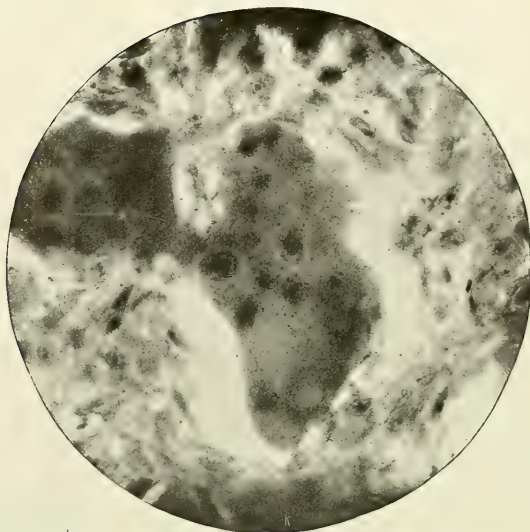
FIG. 304.—Two myeloid cells in a myeloid sarcoma attached to the periodontal membrane and bony socket of a molar.   Magnified 500 times.
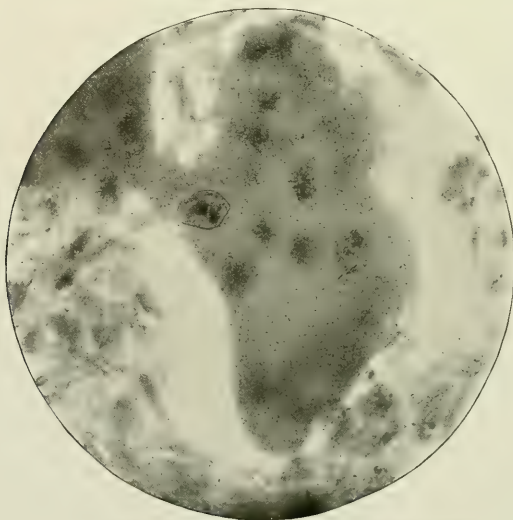
FIG. 305.—The same, magnified 750 times.

These cells vary in shape in different parts of the growth, and contain varying numbers of small nuclei—as many as two or three hundred have been counted—or a few large nuclei or even one large nucleus, showing no evidence of subdivision. They may be vacuolated, and are usually surrounded by clear spaces of different width, the result of rapid shrinkage during the fixing and hardening of the soft tissues (see Figs. 304 and 305).

FIG. 306.—Melanotic sarcoma. Stained with hæmatoxylene and eosine. Magnified 150 times. A. Alveolar stroma containing many round and spindle cells; P. Pigment granules of melanin.

(vii) *Melanotic Sarcoma*

Melanotic sarcoma of the palate may occur as a downward extension from a melanotic sarcoma of the choroid coat of the eye. The rarest of all forms of sarcoma, it is extremely malignant, becoming rapidly disseminated in the lungs and other organs.

It generally consists of small round cells which contain pigment granules. Pigment also exists in the intercellular substance.

(viii) *Endothelioma*

May occur in the buccal cavity, but is very rare. It represents a sub-variety of the sarcomata, but differs from them both morphologically and histogenetically.

Thus the term includes all neoplasms which take their origin in endothelial cells, either those of the blood-vessels of the lymphatic spaces or the lymphatic vessels. It is easy to confuse them with the carcinomata, which in many ways they very closely simulate.

The endotheliomata found in the gum most probably originate in the walls of the blood or lymphatic system.

HISTOLOGY

The bulk of the growth consists of cells, often arranged in alveoli, like the alveolar sarcomata, columns of endothelial cells—round, flat, or cubical in shape—which unite in an irregular fashion with each other, and nests of epitheloid cells. A fair amount of stroma of connective-tissue fibres is present between the cells of the alveoli.

II. TUMOURS OF THE COMPLEX CONNECTIVE TISSUE TYPE

(ix) *Papilloma of the Gum*

GENERAL CHARACTERISTICS

Definition.—An innocent tumour of papilliform character confined to the mucous membrane.

Pathology.—This neoplasm belongs to a papillary type of growth assuming the morphological characteristics of villous excrescences and projecting, as a compound nodular mass above the surface of the gum or mucous membrane of the palate. It is closely related to the condylomata, *verrucæ*, and villous growths of the urinary bladder, rectum, larynx or trachea.

The papillomata vary in size and shape, but often appear pedunculated, and have a tuberous floral aspect. Dense in consistency, they are devoid of tactile or painful sensations, grow slowly, and possess all the signs and symptoms of benign tumours.

Two varieties exist—hard and soft. Of these the hard variety is the only one found in the mouth.

Etiology.—The cause is obscure; but probably long-continued localised irritation is an important factor. Embryonic influences are difficult to trace.

HISTOLOGY

The epithelium is of the stratified squamous variety. Very abundant, it consists of many layers, and presents various stages of corneous transformation.

Fɪɢ. 307.—Papilloma of the palate. Stained with Ehrlich's acid hæmatoxylene. Magnified 45 times.

In length, the longest papilla may vary from 1.8 mm. to 2 mm.

The body of the growth consists of dense connective tissue fibres, of which the cell elements are similar to those of the gum. There is increase in size and alteration in shape of the ordinary papillæ. The vascular supply is more abundant than usual, and the con-

stituent cells and fibres of the part are of a coarser nature than normal.

(x) *Hæmangioma of the Palate*

GENERAL CHARACTERISTICS

Definition.—A tumour consisting of masses of blood-vessels.

Varieties.—Included under the term angioma are:

(i) Nævi or birth-marks.

(ii) Plexiform angiomata, or cirsoid aneurysms, composed of newly formed vessels.

(iii) Cavernous angiomata.

Fig. 308.—Hæmangioma of the palate. Stained with borax-carmine. Magnified 30 times. o.e. Oral epithelium; b. Connective tissue with blood-vessels.

Of these, nævi may be found on the lips; the presence of plexiform angiomata in the mouth has never been recorded; but cavernous tumours may occur.

These are made up of communicating spaces filled with blood and separated by connective tissue in greater or less abundance.

Etiology.—Their origin is unknown.

HISTOLOGY

A cavernous angioma—hæmangioma—consist of numerous rounded or oval spaces enclosed in delicate walls of connective

tissue, and lined with endothelium. The spaces are filled with blood corpuscles, while imbedded in the thin branching connective tissue fibres outside are some escaped leucocytes and proliferated tissue cells. The vessels in places are thrombosed, and hæmorrhages of varying degrees of magnitude take place.

The surface is clothed with epithelium, which has undergone no change from a normal condition.

The process of phagocytosis, induced, in some instances probably, by the death of the blood elements occasioned either by their escape into the surrounding tissues or stagnation in the spaces, has not been observed in any specimens under consideration.

(xi) *Osteoma*

Definition.—A connective tissue homologous tumour arising only in connection with bone, and essentially differing from calcification or ossification of other tumours. Osteomata may be classified as circumscribed or diffuse. The former are found in the mandible, etc., and the latter in the maxillary sinus.

The tissue consists of hard cancellous bone (see Fig. 316). Haversian canals may or may not be present. Periosteum surrounds the new growth.

III. EPITHELIAL TUMOURS

(xii) *Adenoma*

Definition.—A tumour of the type of epithelial or glandular tissue; benign; only arises from pre-existing glandular tissue. Adenomata do not secrete, and have no ducts.

Varieties.—(i) Acinous, (ii) Tubular. The former are found, amongst other places, in the base of the tongue, the lip, the parotid: the latter in the antrum.

HISTOLOGY

Secondary Changes.—Cystic degeneration following on mucoid softening and fatty degeneration of the epithelium.

Cylindrical epithelial cells with a nucleus in the centre of each, in groups, separated by connective tissue with blood-vessels.

(xiii) *Carcinoma*

Definition.—Carcinomata are malignant growths which consist of epithelial cells contained in an alveolar stroma, derived from pre-existing epithelium.

This important group of malignant tumours is anatomically divided into three classes:—

    1. Spheroidal-celled,—*e.g.*, scirrhus, medullary.

    2. Squamous-celled,—epithelioma.

    3. Columnar or cylindrical-celled,—*e.g.*, of the rectum, uterus, etc.

Further sub-divisions, based on pathological and clinical principles, are made.

Of these, however, only one form need demand the attention of the dental surgeon, viz., the squamous-celled genus known as the epitheliomata.

These spring either from the skin of the lips or the mucous membrane of the mouth, which in Vol. I has been described as being covered principally by stratified squamous or "pavement" epithelium.

Etiology.—Long-continued irritation of the mucous membrane of the gum or palate, due to the presence of sharp edges of carious teeth, rough surfaces or margins of ill-fitting dentures, etc.

In the case already quoted on p. 268, the cause could, without hesitancy, be attributed to the irritation of the epithelial "rests" in the periodontal membrane due to septic infection from the pulp of a dead tooth.

Pathology.—In its earliest phases of development a squamous-celled carcinoma is either a small superficial indurated nodule on, or a fissure in the gum. It very rapidly becomes ulcerated, and then there appears an excavation whose edges are raised, irregular in outline, everted, and hard in consistency, whose base is indurated and irregular, whose environing structures are infiltrated with epithelial collections and small-celled masses. The lymphatics which drain the part, infect the lymphatic glands, which become enlarged and hard, and, later on, fixed to the surrounding tissues.

Secondary Changes.—Ulceration of the surface.

HISTOLOGY

The substance of an epithelioma is composed of flat, round, or polygonal cells of varying size, arranged in alveoli, and imbedded in a connective-tissue stroma more or less rich in infiltrated cells.

Two portions of the growth may be recognised and having different characters, clearly distinguished.

In the younger developing part the cells are round and the stroma
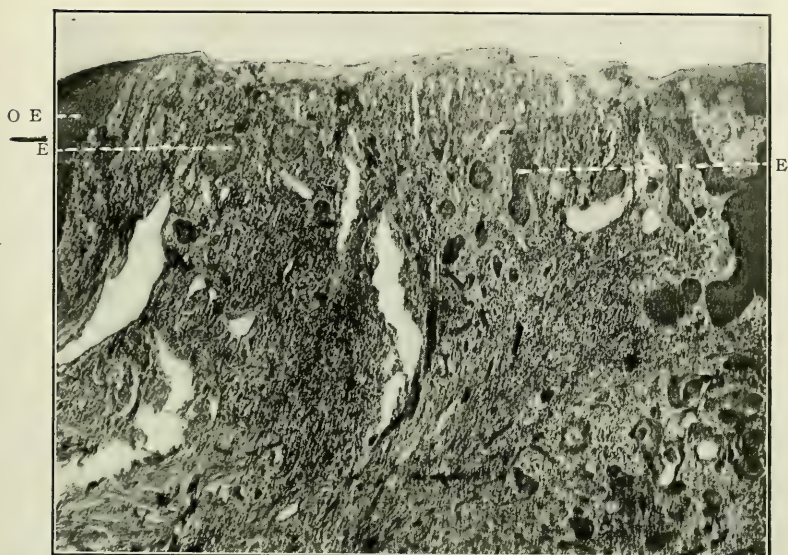
FIG. 309.—Epithelioma of the palate. Prepared by fixing and hardening in alcohol and formalin. Magnified 45 times. Shows ulcerated surface. O.E. Oral epithelium; E. Columns of epithelium extending irregularly into the sub-mucous tissue.
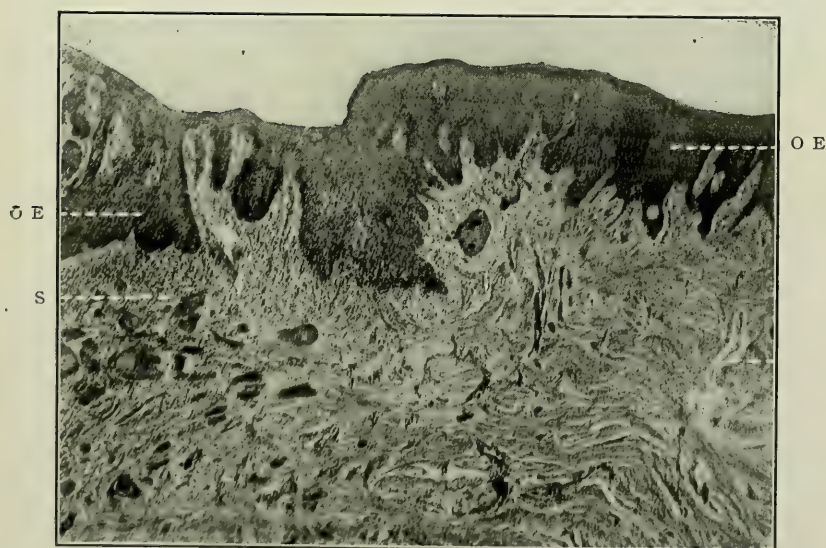
FIG. 310.—Another portion of the same. Shows the epithelial margin of the ulcer depicted in the previous figure. O.E. Oral epithelium; S. Submucous tissue.

extremely cellular; in the older there is a tendency for the epithelial cells to become flattened and to display in places corneous or keratinous changes as in the superficial cells of the *stratum corneum*. Some cells show evidences of great activity in the shape of mitosis.

The epithelial columns which have pierced the basement membrane and have invaded the sub-epithelial tissues are irregular in outline, generally being round or oval. They are discontinuous with the surface epithelium, and, cut off from the oral cells, become isolated and form alveoli or islands of epithelium separated by a
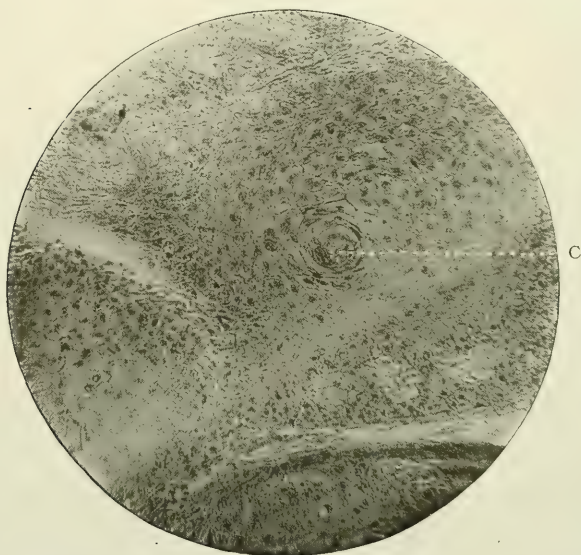
FIG. 311.—Squamous-celled carcinoma of the palate. Prepared by hardening in alcohol. Stained with hæmatoxylene. c. Cell nest. Magnified 50 times.

stroma which contains imperfectly developed connective tissue fibres and small-celled infiltration.

In the epithelial prolongations into the sublying tissues and in the alveoli are often found collections of cell nests.

Cell nests are concentrically arranged whorls of cells gathered around a homogeneous non-nucleated body or group of bodies which consist of keratinous material. This cornification progresses from within outwards, the change represented by the cells being crescentic, cubical and cylindrical respectively.

Cell nests are only pathognomonic of epitheliomata when they

are found in islands imbedded in the depth of the tissues far from the surface with which they are discontinuous.

Some pathologists[1] believe that in the concentric bodies may be found most pronounced evidences of nuclear and protoplasmic
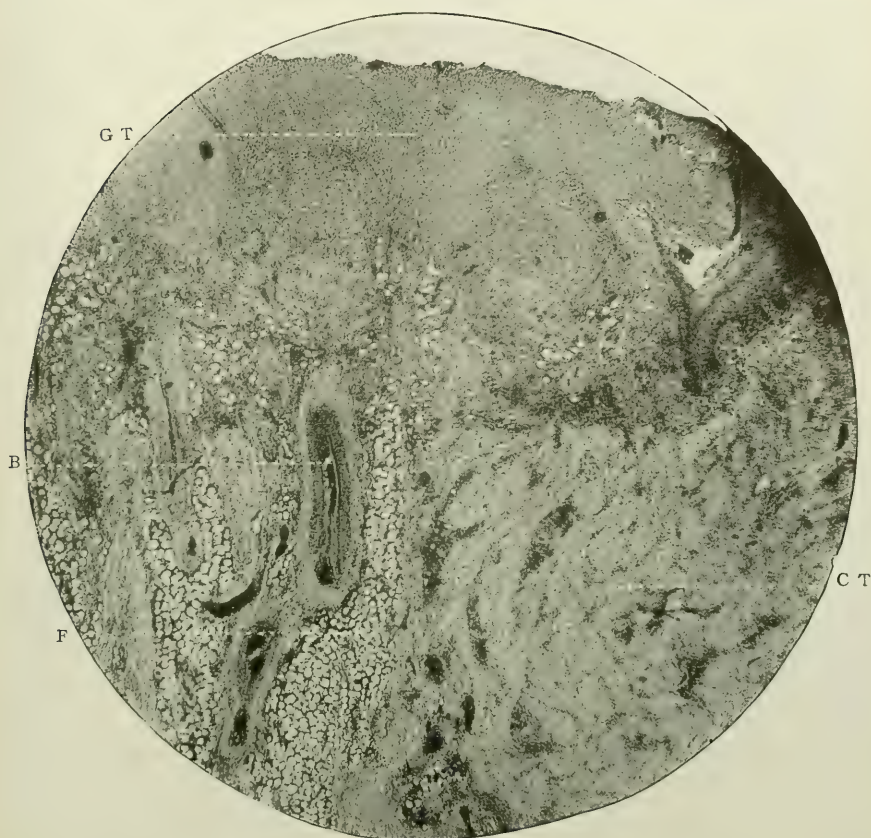
FIG. 312.—A gumma of the mucous membrane of the palate. Stained with hæmatoxylene. Magnified 35 times. G.T. Granulation tissue on free surface; F. Fatty degeneration; C.T. Connective tissue of the palate. B. Blood-vessel cut longitudinally.

necroses, "together with the peculiar process of inclusion of one epithelial cell by another, and an active invasion of leucocytes, some of which are degenerated and others apparently engaged as scavengers to devour the detritus resulting from the necrotic processes."

[1] Hektoen and Riesman. "A Text-Book of Pathology," Vol. I., p. 218, 1901.

Syphilis

This disease may affect the gums and buccal cavity in one of several ways.

Hard chancres of the lips are far from uncommon: and it is possible for these primary sores to occur also on the tongue and gums.
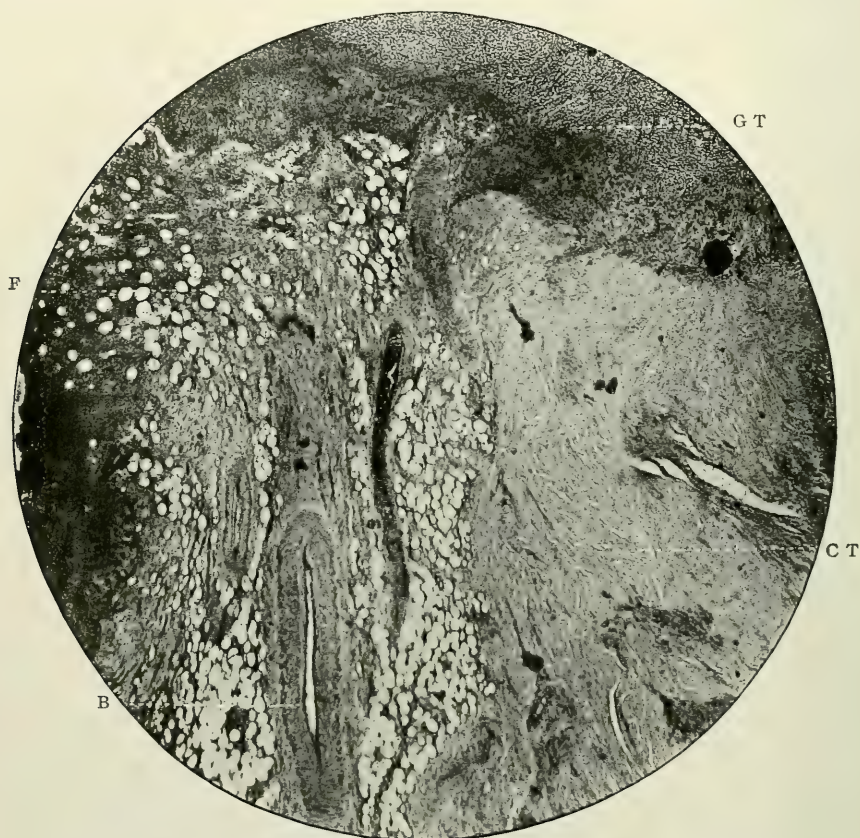
FIG. 313.—The same as the preceding. Lettering as before. Magnified 50 times.

A syphilitic stomatitis often develops in the secondary and tertiary stages of the infection, and gummata may be seen occasionally.

The accompanying photomicrographs show gummatous infiltration of the mucous membrane of the palate.

A gumma is a chronic inflammation which leads to the production of granulation tissue. Varying in size, these lesions, as is seen in Fig. 313, are intimately associated with the surrounding tissues.

They may become absorbed; or remain *in situ* indefinitely without any signs of retrogressive changes; but they often caseate, break down, and form a deep punched-out ulcer.

A gumma consists of a degenerated central part, an intermediate zone of small-celled infiltration, and a peripheral tract or belt of rapidly developing blood-vessels ramifying in a cellular matrix. Fatty degenerative changes are most frequently observed, and, no doubt, are occasioned by the cutting off of the nutritive supply of the most central portions, through changes in the vessel walls, which induce either complete thrombosis or diminution in the calibre of the vessels.

B

OF THE MUCOUS LINING OF THE MAXILLARY SINUS

Inflammation of the mucous membrane of the antrum is similar to that of other soft tissues. Details need not be recapitulated. Suffice it to say that the ciliated epithelium becomes rapidly degenerated and completely disorganised. The cell infiltration is carried on to an enormous extent, the tissue greatly swollen and thickened, and if not suppurative, undergoes fatty and mucoid changes (see Fig. 314).

Spheroidal-celled carcinoma may occur. The photo-micrographs are from specimens given to the author by Sir Francis Farmer. Its chief characteristics are the following:—The cells are spheroidal in shape, and are exceedingly liable to undergo fatty degeneration; they are grouped into large soft alveoli; the separating stroma is very variable in amount, and, when scanty, is devoid of contractile properties. Medullary carcinomata are noted for their abundant vascular supply, in consequence of which hæmorrhages are very frequent in the substance of the growth.

C

OF THE JAWS

The external and internal plates of the alveolar processes of the jaws may, at times, be subject to the formation of osseous growths commonly, though not always correctly, spoken of as exostoses.

Etiology.—Exostoses are very often regarded in the light of tumours, *i.e.*, non-inflammatory growths. But many of the hard nodular periosteal swellings of the jaws may be considered with
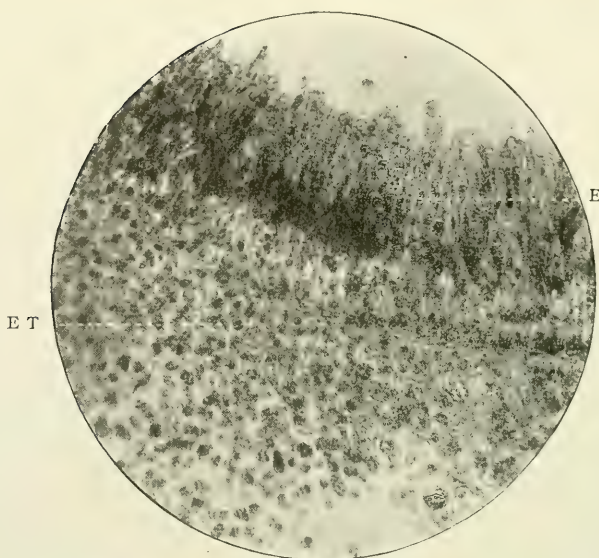
FIG. 314.—Chronic inflammation of the mucosa of the antrum of Highmore. Magnified 230 times. E. Ciliated epithelium; ET. Sub-epithelial tissue infiltrated with inflammatory cells and products. (*From a specimen given to the Author by Dr. Cuthbert Lockyer.*)

FIG. 315.—Hard spheroidal-celled carcinoma of the mucosa of the antrum of Highmore. Magnified 230 times.

equal satisfaction as originating in the form of inflammatory osteo-
phytes or hyperostoses comparable to osteitis in other parts of the
body.   Thus a distinction should be made between circumscribed
osteomata and exostoses.   When occurring in the mandible the
former most likely are due to certain rare developmental anoma-
lies of Meckel's cartilage; when elsewhere, the latter probably
arise either from the periosteum of the jaws or of the teeth.   In the

FIG. 316.—Endosteal osteoma of the mandible.   Stained with hæmatoxylene.
    Magnified 150 times.   B. Bone; M. Degenerated medullary tissue.

first instance, exostoses may be due to a localised periosteal depo-
sition of new bone analogous to the nodes produced by syphilis,
often observed on the surface of the tibiæ, or, as Turner believes,
the outcome of "pyorrhœa alveolaris."

Pathology.—Bony tumours generally appear as sessile swellings
of a dense hard character, covered with thin mucous membrane
which has a normal aspect.   They are painless, slowly growing
tumours, and generally easily diagnosed.

HISTOLOGY

The microscopical structure is interesting. An external shell or crust of compact covers a circumscribed nodule of cancellous bone, which simulates very exactly the normal types of osseous tissue.

Tumours

The tumours most frequently associated with the maxilla and mandible are cystic or solid. The former comprise dental cysts, epithelial and follicular odontomes, and mucous retention cysts (ade-

FIG. 317.—Adenoma of the maxillary sinus arising from the periodontal membrane of a third maxillary molar. Stained with hæmatoxylene. Magnified 45 times. D. Dentine; A. Adenoma; S. Soft tissue attached to the root of the tooth.

nomata) of the antrum, & c.; the latter, growths having the structure of fibrous tissue, mucous tissue, cartilage (enchondromata) and bone (osteomata and diffused and general diffused hyperostoses). Sarcomata and carcinomata, too, unfortunately, are far from uncommon.

It is unnecessary to enter into further details of these various new formations, but brief reference to one of them may be mentioned.

Cystic Adenoma of the Antrum

Cystic degeneration of an adenoma of the antrum of the left superior maxilla, occurring in a woman aged twenty-five is illustrated in Figs. 317 and 318. It had been occasioned entirely by the malposition and fruitless efforts at eruption of the third molar. The periodontal membrane of this tooth had stimulated the mucous antral

FIG. 318.—Cystic degeneration of the adenoma of preceding figure. Stained with hæmatoxylene and eosine. Magnified 250 times. E. Glandular epithelium; C. Cyst filled with albuminoid material; S. Stroma of tumour.

glands to enlarge. The adenoma filled up the greater portion of the upper part of the antrum. In places the bony wall had become absorbed and the new growth extended through the soft tissues and mucous membrane of the palate in the neighbourhood of the molars. In breaking down, a deep punched-out ulcer was produced, which closely resembled a breaking down syphilitic gumma, or an epithelioma.

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CHAPTER XIV

DISEASES OF THE ORAL MUCOUS MEMBRANE

MICROSCOPICAL ELEMENTS IN: (i) Inflammation; (ii) Tuberculosis; (iii) Malignant degeneration.

Stomatitis

Definition.—Inflammation of the mucous membrane of the oral cavity, including that of the lips, cheeks and alveolar process collectively or severally.

DIFFERENTIAL DIAGNOSIS

Variety	Causes	Symptoms	Diagnosis	Terminations or complications
1. Simple catarrhal	(i) *Local:* Faulty oral hygiene, tartar, use of vulcanite dentures, mouth breathing, chemical irritants, abuse of tobacco, etc. (ii) *General:* Dyspepsia, gastritis, gastric ulcer, gout, diabetes, chronic nephritis, infectious fevers, iodism, etc.	Mucous membrane swollen, epithelium thickened, anorexia, fœtid breath, etc. Much mucous secretion.		Favourable.
2. Herpetic	Catarrh and dyspepsia.	Formation of vesicles which soon rupture and leave scabs.	Mouth dry. Removal of epithelium accompanied with pain and hæmorrhage.	Quickly subsides.
3. Aphthous	*Oidium albicans.*	Curdy, easily-detached, white patches.	Mouth always moist, epithelium easily peeled off.	May be fatal through diarrhœa and septic absorption.
4. Ulcerative	Dyspepsia, local irritation, defective oral hygiene.	Grey sloughs, foul breath, gums red and swollen.		Favourable.
5. Syphilitic	Secondary and Tertiary.	Symmetrical: condylomata and mucous tubercles, gummata.		
6. Mercurial	Abuse of mercury.	Foul breath, swollen gums and tongue, profuse salivation, swelling of parotid and submaxillary glands. Loosening of teeth.		Gangrenous ulceration; extensive destruction of tissues, and perhaps necrosis of bone.
7. Gangrenous or *Cancrum oris*	Capillary thrombosis in cheek, perhaps due to Lingard's bacillus.	Foul breath, great tenderness of parts, black indurated slough on cheek, extensive sloughing.		Toxæmia, septicæmia, bronchitis and pneumonia.

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Varieties.—Herpetic, aphthous, catarrhal, syphilitic, ulcerative, mercurial, and gangrenous.

HISTOLOGY

The pathological changes occurring in the tissues are similar in all respects to those observed elsewhere. They do not require a special description.

Fig. 319.—Tubercular nodule in mucous membrane of cheek. Stained with hæmatoxylene and eosine. Magnified 250 times. o.e. Oral epithelium; G. Giant cells; E. Epitheloid and round cells; B. Blood-vessel.

Tuberculosis

Tuberculosis occurring in the mouth is at times of primary origin. More often, however, it follows laryngeal or pulmonary tuberculosis.

Situation.—It is seen most frequently on the dorsum near the tip of the tongue, on the soft palate, on the mucous membrane of the cheeks and on the surface of the tonsils.

Origin.—It begins as a small superficial nodule, and soon breaks down and produces an irregular ulcer with infiltrated edges.

HISTOLOGY

A typical tubercle consists of many newly developed cellular elements. In its centre are found: (i) giant cells, two or more in number, consisting of granular cytoplasm with many peripherally-arranged nuclei. In and around these cells the *Bacillus tuberculosis* can be demonstrated by special staining. Surrounding the giant cells are (ii) numerous endothelial cells—oval, mononuclear bodies about 10μ to 15μ in diameter. (iii) External to these are many round cells similar to lymphocytes from which they are probably derived.

The accompanying photomicrograph may be considered to be fairly representative of the ordinary type of tubercular nodule found in the mucous membrane on the surface of the voluntary muscle fibres of the cheek. (Fig. 319).

MALIGNANT DEGENERATION

INTRODUCTORY

Possessing no ordinary degree of practical utility, or of interest, for the modern dental surgeon as does a study of the degenerative conditions which may affect the root membrane of the teeth of man—one of which formed the subject of Chapter XII—it will doubtless be readily conceded that a condition which must engage the attention of the dermatologist and oral surgeon, as well as of the general student of this volume, is of necessity, and by virtue of its larger bearing on surgery and on the principles of life and death, far profounder in its importance and vaster in its clinical and pathological significance. Such an one is that type of degeneration about to be described.

The former—fibroid degeneration of the periodontal membrane—having a mesodermic derivation, is a local manifestation of a benign condition which, *per se*, remains always benign; the latter, ectodermic in origin, a local manifestation of a benign condition which, as will be presently seen, may become malignant: the first a product and an accompaniment of senility; the second, of youth or middle age; the one a common termination of a common affection, the other an unusual termination of a constantly occurring neoplasm.

A similar type of degeneration, it is perfectly safe to say, has hitherto never been described as affecting the mouth and its contents.

The mucous membrane of the oral cavity is usually very tolerant of the conditions to which it is subject as a consequence of injury by friction from foreign bodies placed upon it. It may, however, become pathologically affected and present a mottled appearance, or be covered by areas of tissue which look like dried blood-clot. Areas may remain for years unchanged, or if inflamed, may break down and ulcerate or undergo malignant changes. Such conditions should always be regarded with suspicion, and diagnoses made between dental, dyspeptic, tubercular and malignant ulcerations.

A precancerous condition as observed on the surface of a pedunculated fibroma may now be described.

HISTOLOGY

It will be convenient, in the first place, in detailing the minute anatomical appearances, which to the pathologist cannot fail to be of the greatest interest, to use the somewhat arbitrary, artificial divisions adopted by microscopists of the present day, as applied to the epidermal covering of the skin and certain mucous membranes of the body. The histo-pathology may therefore be considered under the headings of—

(i) Changes in the stratum corneum including the *stratum lucidum* and the *stratum granulosum;*

(ii) Changes in the *stratum mucosum;*

(iii) Changes in the sub-epithelial tissue or the *cutis vera.*

(i) *Changes in the Stratum Corneum*

The *stratum corneum* extends over four-fifths of the periphery of the growth; where deficient, there is no differentiation of squamous stratified and columnar cells. But this layer presents many departures from the normal type. In some places it and the contiguous strata are insensibly merged into one another; in others, the former is sharply defined (Fig. 320), and then shows as an unusually thin band of flat, narrow, stratified corneous cells with elongated planary nuclei whose karyoplasm is indistinguishable and structureless. There is a marked tendency, here and there, toward desquamation; and the free surface is partially covered with layers of keratin or

kerato-hyalin material, which presents either as a mass of tiny granules or a denser, more homogeneous, partially detached, ground-glass-like matrix. Polychrome methylene blue rendered the contents of these cells and those of the stratum granulosum very prominent, and accentuated, by means of its chemical affinities, the presence, in places, of flakes of eleiden, (Ranvier) or kerato-hyaline. Fig. 322 shows that in places these superficial layers penetrate almost to the corium of the fibroma.
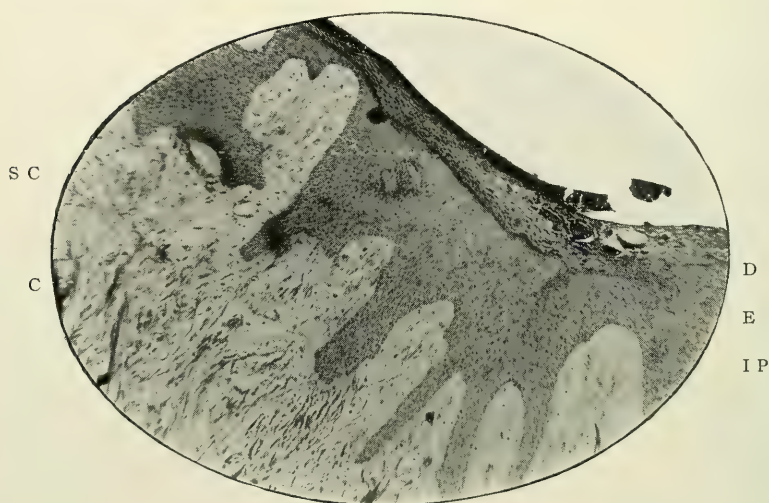
Fig. 320.—Degenerated epithelium on surface of fibroma. Stained with poly-chrome methylene blue and eosine. s.c. Line of demarcation between superficial strata and stratum mucosum; D. Ballooned cells containing kerato-hyaline; E. Epithelial cells of a less degenerate type; C. Connective tissue fibres and cells comprising the substance of the fibroma; I.P. Normal character of interpapillary process of dermis. Magnified 60 diameters.

Particularly striking are irregularly arranged patches of these degenerated cells; they appeared in every section. The cell wall had apparently become grossly distended (ballooning) by retrogressive changes occurring within it (Figs. 321 and 324). The nuclei in many cases are absolutely destroyed; the spongioplasm had disappeared, and the hyaloplasm, which in normal young cells constitutes the greater relative amount of the protoplasm, had evidently become converted into masses of keratinized material which in vertical sections of the epithelium often filled the whole of the ballooned cell and often produced a meniscus-like body, the open space of which was invariably situated at the peripheral portion

of the cell, and sometimes was empty but at others partially filled with granular detritus (Fig. 321). These patches extend some distance toward the centre of the fibroma (Figs. 325 and 326); and the epidermal cells were simultaneously proliferating inward.

(ii) *Changes in the Stratum Mucosum*

A little lower down, and nearer to the *rete mucosum* or *Malpighii*, the cells are more normal in shape and size. The greater number
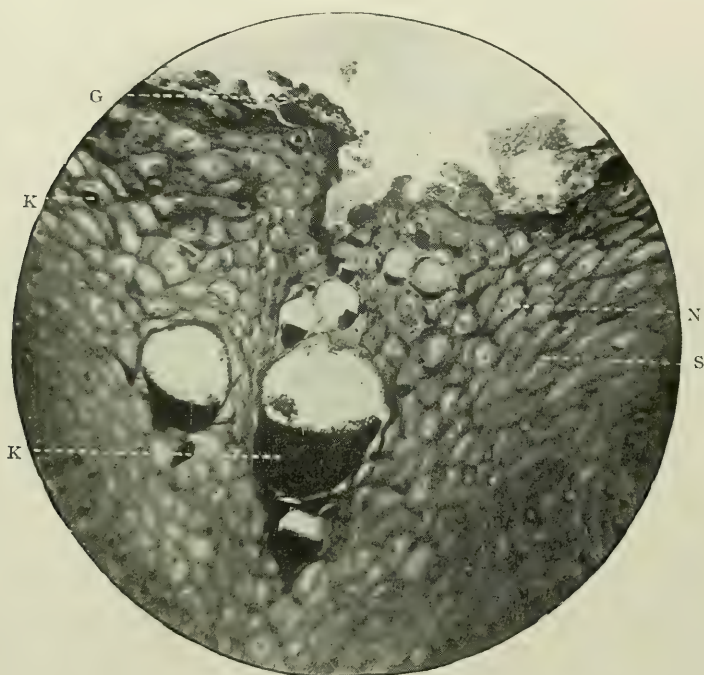
FIG. 321.—To show minuter details of epithelium stained as in preceding. K. Meniscus of kerato-hyaline material; G. Granular type of kerato-hyaline material on free surface; N. Nucleus lying in a clear space; S. Inter-epithelial space devoid of "prickles." Magnified 240 times.

are "prickle" cells; but many have no intercellular bridges and are not separated by interepithelial channels. The majority of the "prickle" cells exhibit their nuclei lying in a clear, narrow space (Fig. 321). Perhaps the greatest interest centres in these nuclei. All changes and rearrangements in their karyoplasm can be noticed. The majority show them in a "resting" stage, but the chromosomes

while large in size seem very few in number—a condition known as
hypo-chromatosis. On the other hand, many of the changes due
to the process of mitosis are observed and the figures well exhibited
in the hæmatoxylene-stained sections. Those treated by the Van
Gieson method are not so brilliant.

Most of the changes familiar to the histologist and pathologist
can be detected. Thus, typical heterotype amphiasters, ring
chromosomes and centrosomes, and many instances of asymmetric
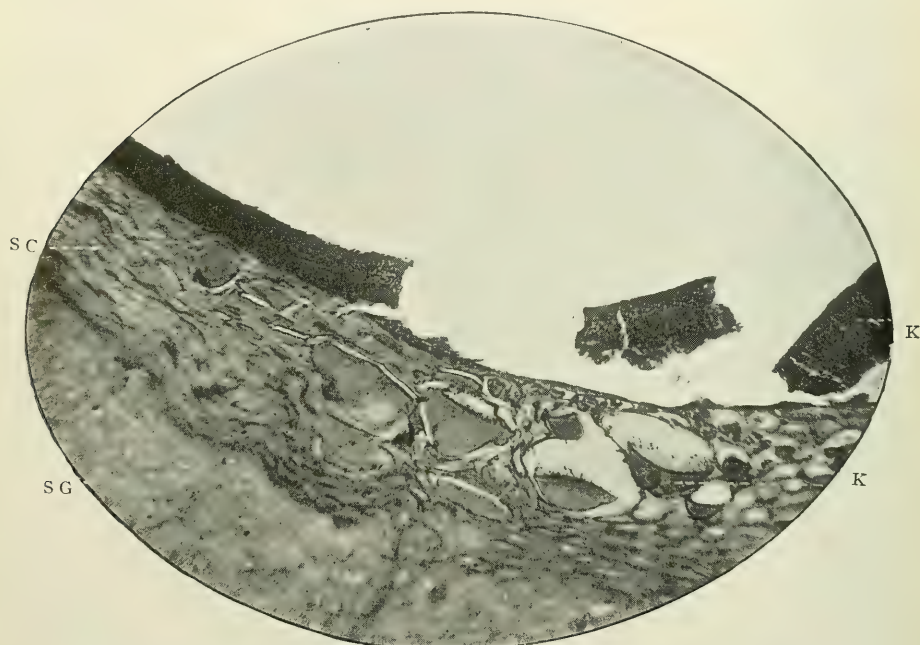
FIG. 322.—Same as preceding, staining and magnification similar. K.
Kerato-hyaline matrix; s.c. Superficial degenerated stratum of cells; s.g. Stratum
mucosum (degenerated).

mitoses occur. There are slight evidences of karyoclasis and karyo-
lysis, but no vacuolation of the nuclei, though the cells themselves
are certainly, generally speaking, undergoing hydropic degeneration.[1]
Darier's "coccidia" and typical myeloid cells are also present, but
not frequent—at least bodies resembling them appear to exist
scattered about in places.

Equally important with the above metamorphoses are the presence
of the typical cell-nests of the squamous carcinomata. A few are

[1] Delafield and Prudden, Pathology, 1901.

discerned near the deepest portion of the *rete mucosum*, and are unduly large, and form, as seen in Fig. 327, a striking feature of the sections.

The delicate basement membrane which supports the *rete Malpighii* is, generally speaking, extremely irregular in outline (Figs. 328 and 329). The columnar epithelial cells situated upon it
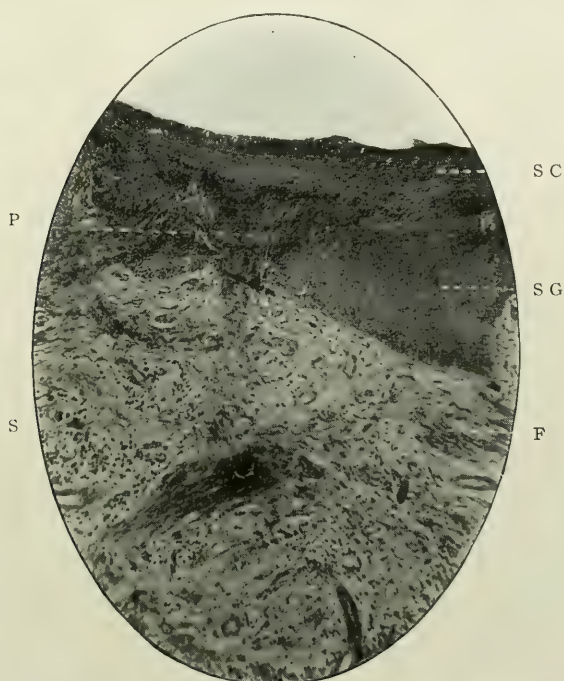
FIG. 323.—Surface of fibroma. Stained with hæmatoxylene and eosine. s.c. Superficial strata of a fairly normal character; s.g. Stratum mucosum with œdematous epithelium; p. Epithelial cells proliferating inward, and breaking through the normal basement membrane; f. Connective tissue of fibroma; s. Small round-celled infiltration. Magnified 50 diameters.

appear to be fairly regular in shape, and to show few evidences of degeneration or œdema. But the uniformity of outline of the basement membrane repeatedly and greatly departs from its usual foldings, and the interpapillary spaces are exceedingly irregular.

In that portion of the periphery of the growth where the *strata corneum, lucidum,* and *granulosum* are absent, the epithelial cells are all more or less œdematous (Fig. 329), and, briefly, exhibit signs of a condition corresponding to eczema of the skin. They are

swollen, rounded, greatly multiplied, and tend to an anomalous degeneration, being absolutely devoid of intercellular bridges. As a result of the colliquative degeneration, vesiculation has slightly occurred; and there are also many migratory cells, etc. Their depth from the surface does not modify these changes (Fig. 330). The process of mitosis is with difficulty made out in these situations, though there is no doubt that it is taking place, and that some proliferation and growth of the epidermis accompanies it.
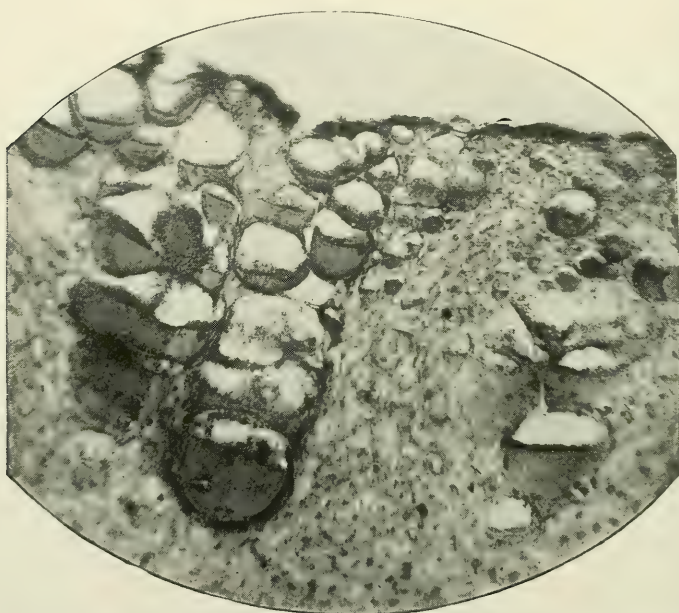
FIG. 324.—Large patches of ballooned epithelium in degenerated œdematous tissue. Stained with hæmatoxylene. Magnified 250 diameters.

(iii) *Changes in the Cutis Vera*

Here the papillæ are seen to be, in parts of the fibroma, very irregular, as already indicated. A noticeable condition is, however, the small round-celled infiltration which has been associated with the cytological metamorphoses. In the neighbourhood of the cell nests, in the most irregularly shaped interpapillary processes, and beneath the œdematous surface this infiltration is most marked. The cells themselves are largely of the Unna's plasma-cell type, mixed with a few leucocytes (polymorpho-nuclear neutrophiles

Fig. 325.—Another degenerated patch showing its superficial position; stained with hæmatoxylene. D. Cluster of ballooned cells; E. Irregular outline of basal layer of epithelium.   Magnified 45 diameters.
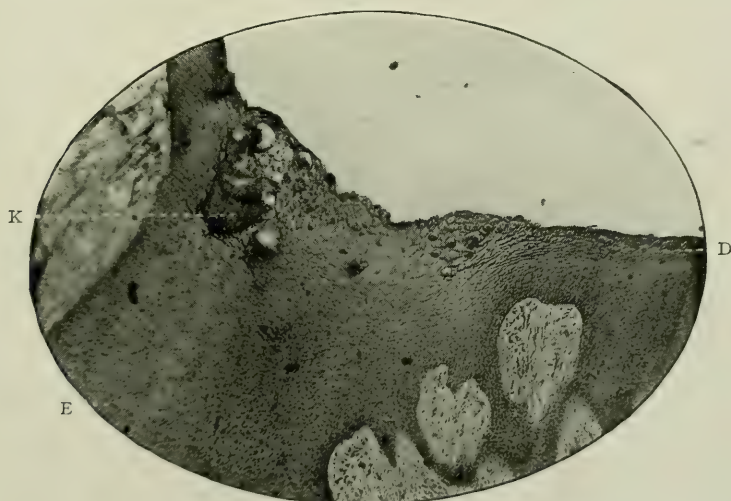
Fig. 326.—Similar to preceding, but exhibiting the various stages in the process of formation of the ballooning of the cells.   Stained with hæmatoxylene and eosine. D. Cells of a fairly normal character, but passing, on the left, to the condition which is at its highest point of development at K; E. Epithelium of stratum mucosum.   Magnified 50 diameters.

and lymphocytes) and some eosinophiles and connective tissue
nuclei and the "mast cells" of Ehrlich. In addition there is an
abundant blood supply.

ETIOLOGY

It is now permissible, necessary, and logically sequential that an
inquiry should be instituted with regard to the theory concerned
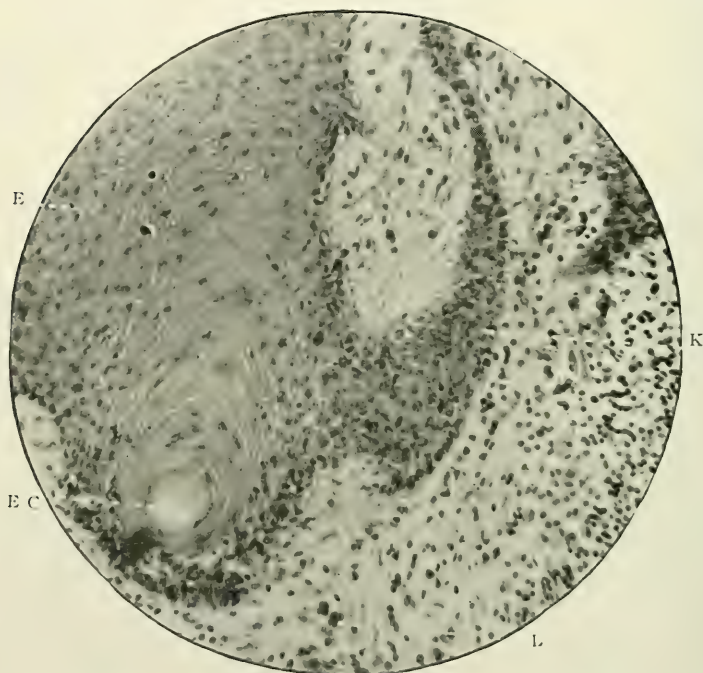
FIG. 327.—Cell nest at base of epithelial column. Stained with hæmatoxylene
and eosine. E. Epithelium of stratum mucosum; E.C. Cell nest showing corneous
character of the central portion; K. Epithelial cells undergoing mitosis; nuclei
displaying asymmetric mitoses; L. Polymorpho-nuclear leucocyte in small-celled
infiltration. Magnified 250 diameters.

in the causation of the changes already described, and an endeavour
made to bring them into line with those of modern research. It
may, however, be stated at the outset that many difficulties beset
this work. The recording of clinical histories and the describing of
macroscopical and microscopical anatomy are accomplished with
facility and some degree of certainty; not so when attempts at
etiology are undertaken.

The exciting cause (or causes) of cancer has for generations per-

plexed the minds of men, and today there is probably no one department of medical science which has more strenuous followers, bacteriology and therapeutics not excepted. It would not be germane to the scope and intention of this chapter if the various questions relative to it were discussed. It must be sufficient if allusion is very briefly made to the latest opinions of authoritative and competent witnesses, and these suggestions applied to the present case.
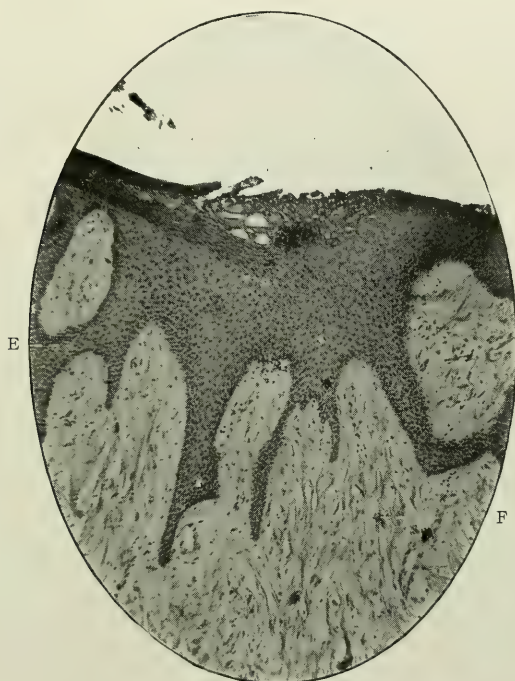
FIG. 328.—Irregularity of epithelial surface. Stained with hæmatoxylene and eosine. E. Epithelium; F. Tissue of fibroma. Magnified 45 diameters.

The clinical notes do not in any way account for the presumably sudden assumption on the part of an innocent tumour of malignancy.

Physical and chemical lesions seem to be at once eliminated as affording the least possible clue. There was no apparent source of irritation or traumatism as occurs unmistakably in mammary scirrhous carcinoma, or labial epithelioma, or "chimneysweep's" cancer, and the predisposing factor of age can also be put out of court. Sections were stained specially to discover if bacterial or protozoan or blastomycetic infection had occurred—with negative results;

although it is most obvious that the skin and mucous membranes generally must be easily subject to invasion by the micro-organisms which they so constantly harbour.

FIG. 329.—Surface of fibroma at junction of eczema-like epithelium, with the epithelial layers of preceding sections. s. Epithelium similar to that in preceding figure; E. Œdematous cells with no superficial strata, and extending in irregular columnar formation into the substance of the fibroma. The rapidly growing epithelium and the round-celled infiltration of the sub-epithelial tissue are shown. Magnified 45 times.

Serious and continuous consideration of the clinical history, combined with the patho-histology, would seem to argue against Cohnheim's embryonal theory, which was originally introduced in 1882, and has been recently revived by Sir Henry Morris, who, in the Bradshaw Lecture of the Royal College of Surgeons of England, in 1903,

strongly advocated the views therein promulgated.   The mechanical isolation hypothesis of Ribbert, as well as the anaplasic theory of Hausemann[1] would also appear to fall to the ground.

But there is apparently nothing to refute, nor, be it remarked, to absolutely verify, the quarter-of-a-century-old doctrines of Thiersch and Waldeyer, who believe that cancer cells take their origin from altered pre-existing epithelial cells.
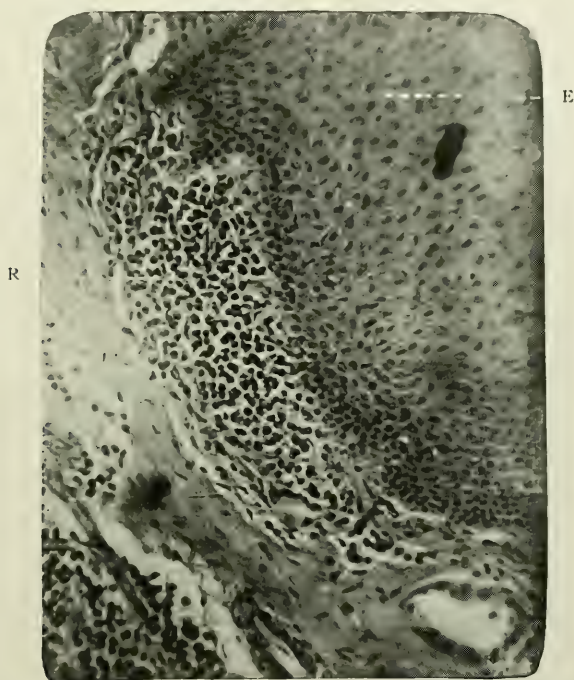
FIG. 330.—Dense cell infiltration of dermal tissue at base of epithelial column. E. Stratum mucosum; R. Cellular infiltration in connective tissue.   Magnified 210 times.

In a most instructive paper presented to the Royal Society of London, in December 1903, by Messrs. Bretland Farmer, Moore, and Walker, entitled "On the Resemblances Exhibited Between the Cells of Malignant Growths in Man and Those of Normal Reproductive Tissues," the following sentence occurs: "In a typical example

[1] See Bashford and Murray, "The Significance of the Zoological Distribution, the Nature of the Mitoses, and the Transmissibility of Cancer," *Proceedings of the Royal Society*, vol. lxxiii, 1904.

of rapidly growing epithelioma it is seen that in the early stages of the proliferation of the Malpighian layer, the cells of the invading tissue at first pass through a cycle of somatic divisions, exactly as in the early stage of reproduction tissue. The resemblance may extend to the frequent production of giant cells, a common occurrence in each case. As cell multiplication proceeds, however, a change passes over the cells themselves. The protoplasmic continuity, to which the "prickly" character is due, becomes more or less obliterated, and the cells assume that appearance of indifferent germ tissue so well known as a feature of the elements of which malignant growths are largely made up."

The conclusions to which these observers came were that malignant new-growths are really nothing more nor less than "reproductive (gametogenic) tissue arising in abnormal situations and possessed of an independence and power of growth like that of the testis in the mammalian body".

The occurrence of the phenomena described by them was corroborated, in the case of the lower vertebrates, by Bashford and Murray, in January, 1904.

To sum up. It may be said that if an explanation were insisted upon for the causation of this disease, the opinions held by Calcott Fox and MacLeod in "A Case of Paget's Disease of the Umbilicus," appearing in the *British Journal of Dermatology*, vol. xvi, No. 2, 1904, taken in conjunction with the so-called "autotoxic hypothesis," might in all probability throw some light on the subject.

The former writers remark, "It has never been demonstrated, and it seems highly improbable, that the degenerated cells which occur in the epidermis or the epithelium in Paget's disease can take on malignant characters. It appears to us, however, more reasonable to assume that the malignant growth originates in epithelial or epidermal cells which, not having degenerated, have reverted and assumed a capacity for proliferation through the prolonged action of some cause which, acting most powerfully on undifferentiated cells, produces their degeneration, than in a matrix of cells which has been deposited there at some period more or less remote." The latter assume that "The products of perverted metabolism in animal organs or tissues produce morbid effects in the individual's body."

In the present instance it is conceivable that the mechanical and physiological growth of the fibroma beneath and internal to its epidermal convering may have induced, in the course of time, the perverted metabolism above mentioned. Although the growth of

the neoplasm was slow, it is entirely probable that it was only quite recently that the epithelium had undergone the carcinomatous changes.

Sir James Paget,[1] who first drew attention in the St. Bartholomew's Hospital reports for 1874 to cases of a certain disease of the mammary areolæ, has been followed by many other contributors to a general knowledge of the subject, among whom may be mentioned Butlin,[2] Wickham,[6] Darier,[4] Duhring,[3] Wile,[5] etc. It has, however, only recently been found that the genitalia may, at times, undergo similar morbid changes, histories having been put on record by Pick, Rolleston,[7] Dubreuilh,[9] Ravogli,[8] and others, including Fordyce, in 1903, at the New York Dermatological Society; while Calcott Fox and Macleod (*loc. cit.*) have reported one instance of Paget's disease of the umbilicus.

The definition of "malignant papillary dermatitis" has been applied to this affection; and it constitutes, in the words of MacLeod ("A Practical Handbook of the Pathology of the Skin," 1903), "a peculiar type of persistent dermatitis which clinically in its early stages somewhat resembles chronic eczema. . . . . In later stages of the disease its malignant character becomes evident, and a carcinoma develops."

CONCLUSIONS

The signification and direct bearing on practical dental and oral surgery of malignant disease of the gums is immediately important and clear. That certain of the innocent neoplasms of the fully formed connective tissue type may undergo secondary malignant changes is a well-known fact in pathology. Thus, lipomata may become sarcomatous, and papillomata and verrucæ carcinomatous. Fibromata usually are subject, if they degenerate or become altered in structure, to calcification and mucoid softening, or ulceration

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[1] "Disease of the Mammary Areola Preceding Cancer of the Mammary Gland." St. Bartholomew's Hospital reports, 1874.

[2] *Medico-Chirurgical Transactions*, 1876.

[3] *American Journal of the Medical Sciences*, 1883.

[4] *Comptes rendus, Société de Biologie*, 1889.

[5] *American Journal of the Medical Sciences*, 1884.

[6] International Congress of Dermatology, Paris, 1889.

[7] *Pathological Society Transactions*, 1897.

[8] International Medical Congress, Rome, 1894.

[9] *British Journal of Dermatology*, 1901.

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when placed on exposed surfaces. It was, probably, a mere accident in the present instance that a fibroma should bear on its periphery signs of malignancy. Epithelia, especially of the pavement or squamous type, are particularly prone to proliferate, and it is a matter of great surprise that the oral epithelium does not oftener produce carcinomata.

The epithelium of the mucous membrane of the cheek would almost seem to possess a large measure of immunity from it. Here there is apparently a frequent source of irritation and tissue change; and yet cheek carcinomata are most uncommon. The surface of lip, tongue, and palate are, however, favourite sites.

The extreme rarity of malignant disease of the gums must not be taken too literally; it may be commoner than is supposed, for it is impossible to say how many fibromata have been excised which were already undergoing these degenerations but which remained unrecognised inasmuch as no microscopical examination had been made.

From the point of view of the frequency of its occurrence it might, perhaps, occupy a position intermediate between that of the cheeks and the other structures named. As the removal of a diseased periodontal membrane will often obviate the formation and development of a dental cyst, or even perhaps sarcoma or epithelioma of the jaws, so too the removal of what is considered to be merely an "ordinary epulis" may prevent the onset of Paget's disease. Extirpation may really signify prevention of its inception.

CHAPTER XV

ODONTOMES AND ODONTOCELES

MICROSCOPICAL ELEMENTS IN:—(i) Epithelial odontomes; (ii) Follicular odontomes; (iii) Radicular and (iv) Composite odontomes; (v) Subcapsular odontoceles; (vi) Extra-capsular odontoceles.

Definition.—"A tumour composed of dental tissues in varying proportion and different degrees of development arising from tooth germs or teeth still in the process of growth"—(Sir John Bland-Sutton). "An odontome is a tumour derived from the special cells concerned in tooth development. . . . . It is a mass of new formation which tends to grow or persist, fulfils no physiological function, and has no typical termination; it also fulfils Thoma's definition of a tumour being 'an autonomous or independent new growth,' the law laid down by him that 'it reproduces with more or less deviation the structure of the part from which it primarily arises.'" ("The Report on Odontomes," London, 1914.)

Classification.—According to Sir John Bland-Sutton,[1] these tumours may be grouped as follows:—

(A) Aberrations of the enamel organ:—
    1. Epithelial odontomes.
    2. Calcified epithelial odontomes.

(B) Aberrations of the follicle (capsule):—
    1. Follicular cysts.
    2. Fibrous odontomes.
    3. Cementomes.

(C) Aberrations of the dental papilla:—
    1. Radicular odontomes.
      (i) Dentomata.
      (ii) Osteo-dentomata.
      (iii) Cementomes.

(D) Aberrations of the whole tooth germ:—
    Composite odontomes.

(E) Anomalous odontomes. (Compound follicular odontomes.)

[1] *Trans. Odonto. Soc.,* Nov. 1887.

This classification has been revised (1914) by a committee composed of Messrs. Douglas Gabell, W. Warwick James, and J. Lewin Payne, and is as follows:—

1. Epithelial odontomes (where the abnormal development occurs in the dental epithelium only):—

    1. Multilocular cysts.
    2. Dentigerous cysts.
    3. Dental cysts.

2. Composite odontomes (where the abnormal development takes place primarily in the dental epithelium, and secondarily in the dental papilla, and may occur in the follicle (capsule) also):—

    1. Complex composite odontome.
    2. Compound composite odontomes.
    3. Geminated composite odontomes.
    4. Gestant composite odontomes, in which a denticle is contained within, or surrounded by, the walls of a tooth, called by Arkövy "*odontoma internum liberum.*"
    5. Enamel nodules.
    6. Dilated composite odontomes.

3. Connective-tissue odontomes (where the abnormal development takes place in the dental tissues of mesodermic origin alone):—

    1. Fibrous odontomes.
    2. Cementomes.

Epithelial Odontomes

Origin.—They may arise from abnormal growth of the epithelial "rests" found in (i) diverticula from enamel organs, or (ii) aborted tooth germs.

Synonym.—"Multilocular cystic tumours."—(Eve).

Macroscopical Appearances.—A soft mass containing cysts of various sizes lying between the external and internal alveolar plates which have become much attenuated and enlarged (Figs. 331 and 332). A more or less completely formed tooth can generally be found in the neighbourhood of the growth. At times portions of the tumour may become ossified, columns of bony-like substance being embedded in its midst; or the whole growth may undergo osseous changes. Such cases have been described by Tomes and Miller.
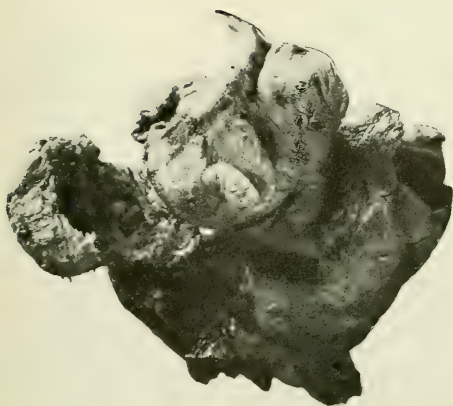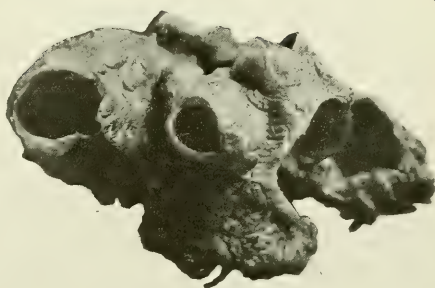
FIG. 331.   FIG. 332.

FIG. 331.—Lateral view of an epithelial odontome. Actual size.
FIG. 332.—A superficial aspect of the same showing several cysts.
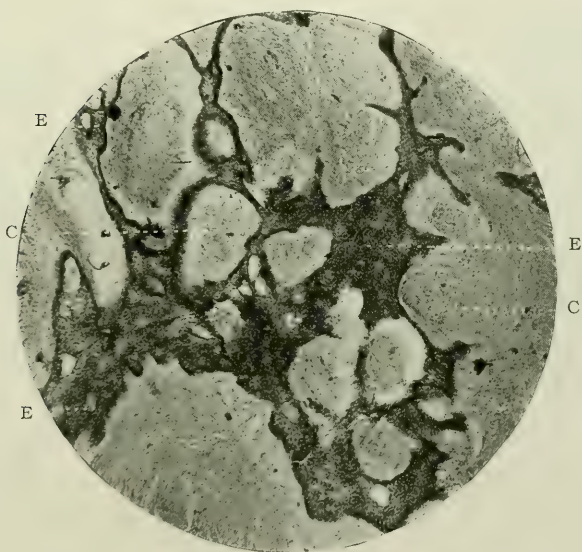
FIG. 333.—A section through the thickest portion of the mass, showing the epithelial columns extending into the mesodermic tissue. Prepared by hardening in alcohol. Stained with hæmatoxylene. Magnified 45 times. E. Epithelium; c. Connective tissue.

HISTOLOGY

The mass chiefly comprises bundles of connective tissue fibres, densely and closely packed; in places a round celled infiltration, which probably represents embryonic mesodermic tissue, can be observed.
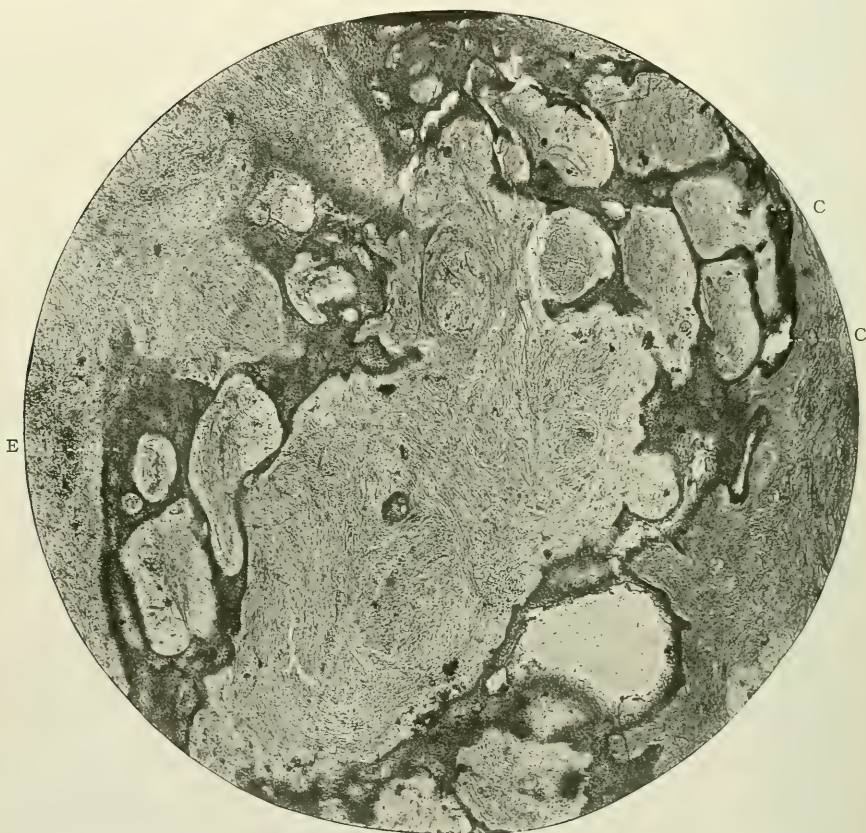
FIG. 334.—Similar to the preceding. Magnified 50 times. c. Early stages in the formation of a cyst; E, Epithelium.

Greater interest, however, centres in the epithelium. This extends primarily in the form of solid rods or territories into the substance of the odontome. The epithelial columns anastomose and branch freely. In some situations they are extremely narrow, in others broad. Thus they vary in width from 0.6 mm. to 1.0 mm.

FIG. 335.—Similar to the preceding. A longitudinal section through the entire wall of a small cyst, showing its contents. E. Epithelial tissue; C. Cyst contents; M. Mesodermic tissue.
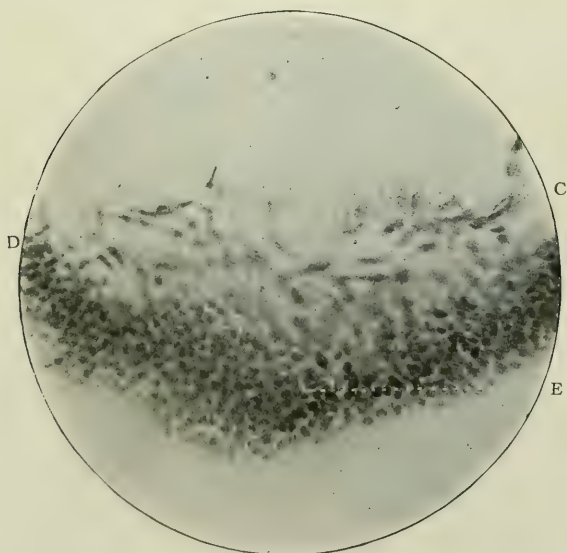
FIG. 336.—A portion of the wall of a cyst in an epithelial odontome. Prepared as the preceding. E. Epithelial cells; D. Degenerate cells; C. Colloid material which constitutes the cyst contents.

Islands of mesodermic fibrous tissue are often formed by the junction of the under side of the epithelial involutions.

The cells here are very similar in constitution to those of the deepest layers of the oral epithelium. In shape they vary somewhat. Those at the margins of the ingrowths are small and closely packed together, each having nuclei, with pronounced chromatin granules and karyoplasm. They are cubical in shape. Those in the central portions of the mass are branched, and have elongated flattened nuclei, many of which contain nucleoli in addition to the karyoplasm. Many signs of rapid subdivision of the cells can be noted. In the centre of the earliest-formed islands the method of breaking down or dissolution of the cells, with the first commencement of cystic degeneration, can be easily studied. They assume a shrunken appearance, with hydropic nuclei. Joined by their processes, they bear a distant resemblance, as Bland-Sutton has suggested, to the stellate cells of the enamel organ.

Follicular Odontomes or Dentigerous Cysts

Origin.—An excessive amount of secretion of fluid between the enamel or Nasmyth's membrane and the dental capsule, which, accumulating, distends the space and produces a cyst.

Synonym.—Dentigerous cyst.

Contents.—A glairy fluid, with crystals of cholesterine and broken down epithelial cells in suspension, and a fully or partially developed tooth or teeth lying in its interior.

They may be (i) Simple (containing one tooth) or (ii) Compound (containing many teeth).

GENERAL CONSIDERATIONS

Teeth found in these cysts usually possess no Nasmyth's membrane. The recognition of any new fact is likely to be succeeded by the advancement of some novel theory which may serve a useful end in attempting to explain what is otherwise inexplicable. Such is the difficulty of obtaining reliable, and therefore, scientific information on many obscure physiological and pathological processes, that occasionally the most commonplace and minor observation becomes the progenitor of a vast store of valuable knowledge.

These generalisations apply equally to the various regions of the body, and to every organ or tissue which they contain or of which

they are constructed. If etiological or pathological processes were more frequently contemplated from the anatomical standpoint, there would be less confusion as to the manner of operation of morbid changes, and as to the successful and non-empirical treatment of diseased conditions.

It would seem at first sight a somewhat trivial and unimportant matter that the retained teeth of dentigerous cysts may have no Nasmyth's membrane investing their coronal surfaces, or, indeed, any portion of them. But it is probably true. Some time ago the author removed from a dentigerous cyst a tooth so affected.

In the mouth of a patient, a boy of nine and a half years, there existed a swelling in the neighbourhood of the left mandibular second deciduous molar. After operation, the second premolar was easily detached uninjured from the floor of the cavity on which it was lying horizontally. It was thereupon subjected to Paul's phloroglucin and nitric acid mixture, in order to ascertain if the enamel cuticle was intact or diseased. It was found to be entirely absent.

The rarity of this form of cyst, or follicular odontome, according to Bland-Sutton, and the heretofore incorrect statements as to the nature of the membrane, have prevented much, if any, investigation of the subject. Although at present the author has been fortunate enough to obtain one specimen only, it is, however, extremely probable that it is the rule, that when dentigerous cysts occur there is concomitant absence or failure of development of Nasmyth's membrane.

To what theories does this fact, then, give rise?

The anatomical relationships of the parts around an unerupted tooth must be briefly recalled. Beginning from within, and passing outwards, the following structures meet the eye, and are placed in order, as—the external free surface of the enamel, the translucent pellicle of Nasmyth's membrane, its cellular layer, the internal layer of the dental capsule, and the external layer of the same.

The cyst wall consists of "dense strands of longitudinally arranged connective-tissue fibres, which are thick and strong, but loose in texture nearest the bone; while gathered into bundles further in, composing the bulk of the cyst, are connective tissue fibres and cells, all freely supplied with blood-vessels. . . . . Ultimately a fully developed dentigerous cyst is lined with a layer of epithelial cells, one or more deep." Like all cysts of new formation. Whence comes this layer of epithelium? It is probably identical with the

external epithelium of the enamel organ, which, on completion of development, becomes the cellular layer of Nasmyth's membrane. If this is the case, then the cyst contents are probably formed by the degeneration and liquefaction of the *stellate reticulum*.

Inspection of a tooth germ at an early stage of growth, when cut in a vertical position, reveals the *stellate reticulum* of the enamel organ bounded externally by a layer of small round cells (external epithelium), internally by round or polygonal cells (*stratum intermedium*), and cylindrical cells with prominent nuclei (internal epithelium or ameloblasts). The first-named, at one time, extends without interruption almost around the entire tooth germ. The actual date of its disappearance is unknown. It is clear, however, that that portion situated near the cusps or incisive edges of the teeth is the last to atrophy.

Of all the structures in the enamel organ which could possibly originate the thick, glairy fluid which forms the contents of the cyst, the *stellate reticulum* is the one. As has been already mentioned the external epithelium probably remains as the internal lining of epithelial cells in the fully developed cyst wall. It has, in this instance, been modified as far as its functions have been concerned, and has never formed the cellular layer of the enamel cuticle, for that thin film has never existed. It is impossible to conceive that, had it been present, it had been destroyed by any pressure or chemical action on the part of the cyst contents.

The internal epithelium cannot presumably take part in the degeneration. For its work is accomplished in the formation of the enamel, finally, in an atrophied or exhausted state, losing its cellular identity, and degenerating into the keratinous homogeneous pellicular layer of the membrane.

There remains, therefore, the *stratum intermedium* and the stellate cells. Of the former little is known concerning either its function or its real anatomical relationships. The author ventures, therefore, logically and reasonably to believe that it is the cells of the "packing material" which are the all-important factor.

One argument against the acceptance of this view which might undoubtedly be raised is that the *stellate reticulum* disappears very early in the development of the tooth. This is true. But there can be no objection to the expression of the opinion that a portion of it may escape atrophy, and, remaining behind as an unabsorbed body, have its constituent cells suddenly undergoing centrifugal proliferation, with its inevitable termination of degeneration, death,

and liquefaction. The fluid thus produced would have no difficulty in surrounding the whole surface of the crown, and, if the roots were completed, enveloping the entire or greater part of the tooth.

Still it is not easy to prove the anatomical origin of a dentigerous cyst in the light of present knowledge. Nevertheless, one or two considerations may be applied to its study. Thus:—

(a) There is an analogy in the formation of epithelial odontomes, which are believed to arise from irregular diverticula from or unatrophied remnants of the tooth band.

(b) There is a further similarity between the microscopical appearances of the cells of an epithelial odontome which are about to break down and the cells of the *stellate reticulum* as Sir John Bland-Sutton[1] has already noticed ("Tumours Innocent and Malignant," 4th edition, 1906), especially when they are becoming aged.

(c) The teeth found in follicular odontomes are often only partially complete. This agrees with the fact that the *stellate reticulum* disappears early in the life-histories of teeth.

(d) And, lastly, by a process of elimination it is satisfactory to surmise that no other constituent of the enamel organ could produce the cyst contents than these cells.

These remarks are based on the assumption that some cells of the enamel organ play an important part in the genesis of a dentigerous cyst. There is just, however, one other possibility if this suggestion is incorrect, and that is that the follicle itself may originate its own cyst.

Sections of the dental capsule, when made at the time of its fullest growth, exhibit many winding tube-like bodies composed of epithelium, which run in an inward direction, and are almost exclusively confined to its inner border, *i.e.*, the part which is contiguous with and closely applied to, Nasmyth's membrane.

Now, it is possible, but unlikely, that proliferation and growth occur in one or more of these bodies. As these are placed externally to the external epithelium of the enamel organ, one would expect to find the enamel cuticle intact on the surface of the tooth embedded in the cyst. But this is not so.

Hence it is that, taking all things into consideration, and weighing carefully the *pros* and *cons*, the author ventures to express his belief in the origin of the cyst in the manner already detailed,—a belief confirmed by the fact that Messrs. W. A. Maggs and Pare also

[1] "The central cells degenerate and give rise to tissue resembling the stellate reticulum of the enamel organ"—p. 228.
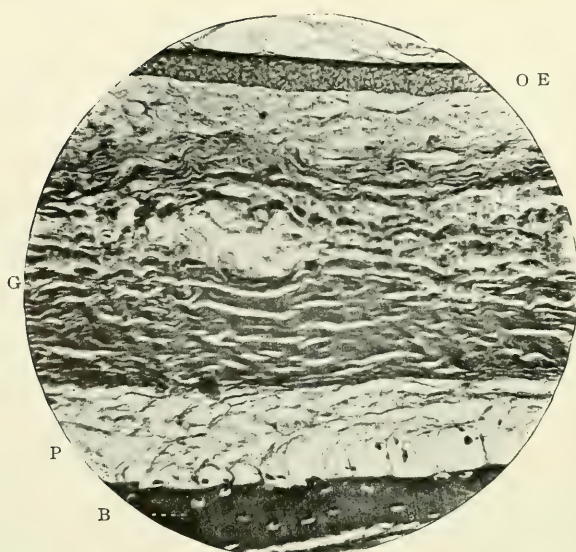
FIG. 337.—The mucous membrane and soft tissue external to the wall of a follicular odontome. Prepared by hardening in alcohol. Stained with Ehrlich's acid hæmatoxylene. Magnified 230 times. OE. Oral epithelium; G. Tissues of the gum; P. Periosteum; B. Attenuated bone.
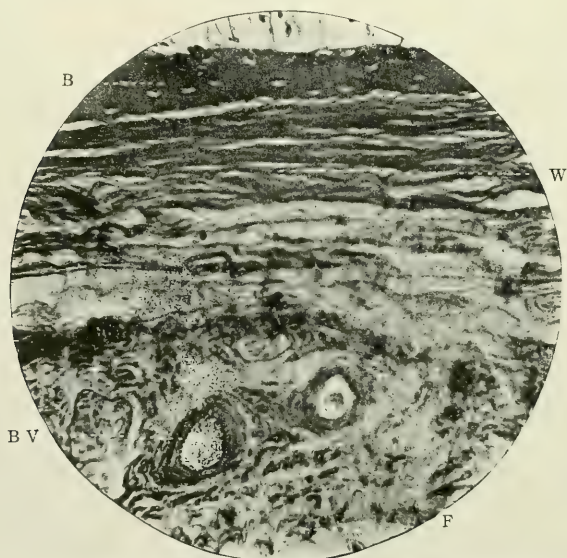
FIG. 338.—The wall of a follicular odontome. The innermost part of the section in Fig. 337. B. Attenuated bone; W. Wall of cyst; BV. Blood-vessels; F. Internal surface of wall.

noticed the absence of Nasmyth's membrane under similar condi-
tions, and noted the anomaly in *Guy's Hospital Gazette* for 1904.

Follicular odontomes are undoubtedly congenital or develop-
mental in their origin. But a good deal of confusion still exists
as to their pathogenesis. It is unnecessary to enter into the argu-
ments of many writers. Let it therefore suffice to recall that,
among the older ones, Broca believed that they arose within the
tooth capsule, sac, or follicle, the enamel organ having disappeared
"under morbid influences;" that Malassez explained that they
were due to hypertrophy of epithelial rudiments of the enamel
organ; that Albarran considered they were inaugurated by the
proliferation of the aborted epithelium of the fibrous tissues normally
present in the *iter dentis;* and that Salter ascribed to "a sort of
epithelium" clothing the so-called enamel pulp "the power of
assuming the function of secreting fluid."

Bland-Sutton defines them thus: "Swellings [which are] often
called dentigerous cysts, a term which has come to be used so very
loosely that it should be discarded in the necessity for precision.
They arise commonly in connection with teeth of the permanent set,
and especially with the molars; sometimes they attain large dimen-
sions and produce great deformity, especially when they arise in the
upper jaws and happen to be bilateral. Rarely they occur in
connection with supernumerary teeth. The wall represents an
expanded tooth-capsule. . . . . The cavity of the cyst usually
contains viscid fluid and the crown or the root of an imperfectly
developed tooth. Occasionally the tooth is loose in the capsule,
sometimes inverted, and often its root is truncated; exceptionally
the tooth is absent or represented by an ill-shaped denticle. The
walls of the cyst always contain calcific or osseous matter; the
amount varies considerably."

Tomes and Nowell write: "The follicular cyst arises in con-
nection with teeth retained in the jaws, generally premolars or
molars. They cause great distension of the jaws" (p. 725); and,
in accounting for their mode of formation and generation of the
fluid contents, assert (p. 732): "When the development of the
enamel is completed, its outer surface becomes perfectly detached
from the investing soft tissue, and a small amount of transparent
fluid not uncommonly collects in the interval so formed."

It is a well-known fact that the deciduous and permanent teeth,
when about to erupt, may present over their crowns a bluish, soft
vesicle containing serum (eruption cysts). It is not an easy matter

to explain the presence of the vesicles over the *teeth of succession* because of the presence of the absorbent organ, and nearly always follicular odontomes are formed in connection with the members of the permanent series. The authors just cited believe that this fact—viz. the occurrence of vesicles—furnishes an explanation of the manner in which cystic tumours containing unerupted teeth arise. "Fluid collects between the enamel and the tooth capsule," they say. Assuming this statement to be correct, and although no mention is made of the enamel cuticle, one is led to ask "Whence comes the fluid? Why does it collect?" And one is told that fluid is normally and universally present over the crowns of unerupted teeth. If this were so, one would expect to find that follicular odontomes were extremely common, which they are not, and that unerupted teeth could never remain in an innocuous state in the bones of the jaws, which they do.

Paul, writing in 1894, observed: "On dissecting them (*i.e.*, the teeth of sheep and monkey in their sacs), it appears that at a certain stage the crown of the tooth was quite free inside the sac, but that at a slightly later stage the sac adhered to the tooth, although it could be easily stripped from it."

The above named authors proceed: "As the cyst enlarges, the contiguous bone is removed to make room for it, fresh bone being concurrently deposited on the outside of the jaw. In the case of such cyst lying in front of a tooth which is being cut, it is obliterated by the advancing tooth or it bursts; but when situated deeply in the jaw, a cystic tumour may be the result."

Finally, Heath defines follicular odontomes as "cysts [which] contain one or more teeth in their interior or in their wall. The teeth may be well formed or may be quite rudimentary, consisting of irregular masses of bone and enamel."

The Origin of the Cystic Fluid

Already the opinion has been expressed that the fluid contents of these tooth-bearing cysts is derived from the degenerated cells of the stellate reticulum of the enamel organ. Instead of becoming absorbed in the usual way, they may, for some recondite reason, not disappear, but may further degenerate and liquefy, and produce a potential cavity which, on being filled with fluid, is the beginning of the formation of the cyst.

General Observations

As bearing on the pathology of these cases, it is necessary to recall as succinctly as possible the anatomical topography of the parts, and to explain the grounds for the belief that the liquefaction of the stellate reticulum would account for the fluid in a dentigerous cyst. The enamel organ at maturity consists of four different cell elements, arranged from within outwards, as the (1) internal epithelium or the ameloblasts— elongated, columnar cells, measuring 15μ to 20μ, set in immediate apposition; (2) the *stratum intermedium*, a narrow layer of small polygonal cells; (3) the stellate reticulum, mucoid cells with round nuclei and numerous long branching processes; and (4) the external epithelium, a layer of single rounded or flattened cells. The functions of these are supposed to be as follow: The first to form enamel, and, when "spent," the translucent pellicle, or inner layer of Nasmyth's membrane; the second to recruit or rehabilitate the ameloblasts; the third to act as a "packing" material to the enamel organ; and the last (somewhat doubtful, but believed by some—Professor Paul, for instance) eventually to constitute, on persistence, the cellular layer of the enamel cuticle. Now, outside this external epithelium, which, it is important to note, is in direct continuity with the fibrous tissues which form the dental capsule— there is no sharp line of demarcation between the two, though in speaking of the two structures one unconsciously dissociates them— comes the capsule or tooth-sac itself. Composed, *when young*, of fibrous tissue with a feeble supply of round cells, it contains numerous gland-like epithelial bodies.

It is therefore clear that the dental capsule usually has, on its internal aspect, a layer of epithelium—viz., the external epithelium of the enamel organ. It is possible, in fortunate circumstances, to show the lining of the capsule. To give a concrete example: If an unerupted first premolar be removed at the age of 7 to $7\frac{1}{2}$, and its soft-tissued investment allowed to remain *in statu quo ante*, on making vertical sections one can see, under the 4 mm. objective, the ameloblasts becoming converted into the translucent pellicle of Nasmyth's membrane. At the cervical margin of such a tooth they are elongated and cylindrical, but little altered from those of activity— merely shorter. Higher up, nearer the cusps of the crown, however, they are shrunken and flattened, and while still retaining their prominent nuclei, often become hexagonal or pentagonal in outline. In the neighbourhood of the extremities of the cusps they have

become fused to form a homogeneous membrane—the pellicular or inner layer of Nasmyth's membrane. Next to them is a single flat layer of cells, the external epithelium closely applied to their surface. Though so intimately approximated there is a wide range of difference from an embryological point of view between the two, the external epithelial cells being ectodermic and the dental capsule mesodermic in origin.

Now, if the stellate cells of the enamel organ undergo further softening, disintegration and liquefaction, the first stage of the formation of a follicular odontome is inaugurated. Little by little the fluid collects, until a cyst is produced, with the external epithelial cells on the outside and the internal epithelial cells on the inside. The first form a definite layer of epithelial cells and are not secretory cells. The second has sometimes not had an opportunity of becoming metamorphosed into the pellicle of the enamel cuticle (this is quite conceivable, and, really, a common-sense view) on account of the accumulation of the fluid; hence its absence on three authenticated occasions. At other times the fluid has probably collected *after* the production of the pellicle had occurred, but the cellular layer, instead of appearing on the surface as part of Nasmyth's membrane, remained as the adventitious lining of the cyst wall.

Another fact in agreement with the opinion which is being postulated is the striking anatomical resemblance between the degenerate cells of the stellate reticulum and the degenerating cells of an epithelial odontome.[1]

The majority of the epithelial bodies, derived from the fenestration of the tooth band, may give rise to the eruption cysts or epithelial odontomes, supernumerary teeth, etc.

It is also conceivable that for some reason or other the cells of one or more of these gland-like bodies might undergo karyokinesis, and, like the paradental "rests," though in consequence of a dissimilar stimulation, might rapidly multiply, form large epithelial masses, of which the central cells, cut off from their nourishment, would die, degenerate, and liquefy. Here, then, a cyst might form—a capsular cyst—but if it were evolved from the central portions of the dental

[1] The phenomena associated with this degeneration were thoroughly described by Eve in an important paper on "Cystic and Encysted solid Tumours of the Jaws, with Observations on the Structure of the Enamel Organ," which he read before the Odontological Society of Great Britain in 1885. From his remarks it was apparent that he had corroborated the results of the researches of Falkson and Bryck of Germany, who had independently arrived at similar conclusions.

follicle, there would ultimately be found two layers of heteromorphic cells in the perfected follicular odontome wall; and this is never so. A follicular odontome is therefore an example of a congenital anomaly.

HISTOLOGY

Sections through the entire wall of a follicular odontome reveal most externally, an exceedingly thin layer of the mucous membrane of the gum, the epithelium of which is not more than a dozen cells deep. The outermost are flat, with compressed nuclei; the inner are cubical, as usual.

Below the oral epithelium the fibrous tissue of the substance of the gum and the periosteum of the bone are found.

The bony wall enclosing the tumour, and expanded by its development, has necessarily become very attenuated. The measurements of the diameters of the bone and gum tissue vary from 0.1 mm. and 0.3 mm. respectively.

The cyst wall proper, which may have a dimension of 0.5 mm. in width, lines the inner surface of the bone, and consists of dense strands of longitudinally arranged connective-tissue fibres, which are thick and strong, but loose in texture nearest the bone; while gathered into bundles further in, composing the bulk of the cyst, are connective-tissue fibres and cells, all freely supplied with blood-vessels. The former interlace in every direction, and sometimes are so thickly placed together as to present the appearance of longitudinally cut muscle fibres. The latter, of large size, run in opposite directions to those of the fibres.

A thin layer of epithelial cells exists on the most internal aspect of the cyst wall forming its direct lining.

The central cells cut off from all nutritive sources soon atrophy, die, and become disintegrated, and a cleft is formed. Cystic fluid is then produced.

Ultimately a fully developed follicular odontome is lined with a layer of epithelial cells, one or more deep. The contents stain indifferently, consisting microscopically of an amorphous material. From pressure of the accumulating materials the cells become flattened, except those on the extreme edge of the cyst.

Fibrous odontomata (endosteal fibromata) are merely hypertrophies of the outer portion of follicular odontomes; and Cementomata are either an (i) ossification of fibrous odontomata, or (ii) overgrowth on the part of the osteoblasts in the root membrane. Such probably was the origin of the cemental nodule described on page 80.

25

Compound Follicular Odontomes

These are extremely rare in man; but cases have been recorded in the horse and goat. Sir John Bland-Sutton considers that they arise from the dental capsule, the osseous masses which are a characteristic feature being the result of sporadic ossification of the fibrous tissue composing this structure. He found five hundred denticles in the right antrum of a girl aged 11, and Ward Cousins (*Brit. Med. Journ.*, June, 1908) one hundred and nine in the region of the second right mandibular molar in a boy aged 11. The latter partook of the nature of masses of hyperplasic cementum.

An interesting case has been recorded by Bland-Sutton. An antral tumour of two years' growth, in a patient aged 11 years, was found to contain soft vascular tissue, inclosing a vast quantity of bony particles. More than five hundred such pieces were counted. Microscopical examination of the particles showed their structure to approximate to that of cancellous bone identical with the alveolus of the jaws.

Radicular Odontomes

Origin.—An aberration of the dentine germ, being therefore confined principally to the root portions of teeth.
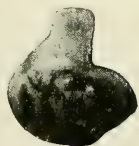
FIG. 339. FIG. 340.

FIG. 339.—Radicular odontome; lingual aspect. Actual size.
FIG. 340.—Radicular odontome; mesial aspect. Actual size.

Macroscopical Appearances.—As types of these and composite odontomes, the two following cases recently described by Dolamore,[1] may be cited.

Occurring in a patient of 14 years of age, a large "flattened enamel-covered tooth-like structure" was found in the region of the right mandibular lateral incisor. It had a second conical mass fused to its lingual surface, which ended below in a smooth bulbous tumour (see Figs. 339 and 340). Its weight was 2.7 grammes. Its measurements were 1.2 cm. in the anteroposterior, 1.9 cm. in the vertical, and 1.6 cm. in the lateral diameter.

[1] Journ. Brit. Dent. Assoc., 1902.

It is difficult to convey by means of mere words or illustrations a precise account of the structure of the conglomerate mass of a tooth tumour; the reasons being, that the hard tissues are so inextricably confused and present so many varied histological appearances which are almost indescribable. At the best of times, therefore, the patho-histology can be but feebly interpreted.
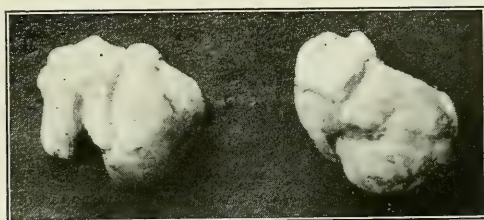
FIG. 341.—Two radicular odontomes.

Sagittal sections having been made, it was found that the incisor, in the case under consideration, is remarkable for the dense pigmentation of its enamel; the brown striæ of Retzius are immature, while the lines of Schreger are altogether wanting. The tooth itself is long. Immediately behind it, *i.e.*, on its lingual aspect, appears

FIG. 342.—Radicular odontome of an incisor or maxillary canine.

another smaller cone-shaped tooth with thick enamel, and a narrowed occluded pulp chamber. More internal still is a third dome-shaped denticle, the enamel of which is fused with that of the preceding (Fig. 343). The dentinal system is absent, the centre of the denticle being composed of a large amount of amorphous or irregular osseous material. Following this, and forming the remainder of the upper and inner surface of the odontome, are several masses of

enamel intimately fused, the innermost one of all resembling a nar-
row cone with an arrangement of dental tubes. The extreme
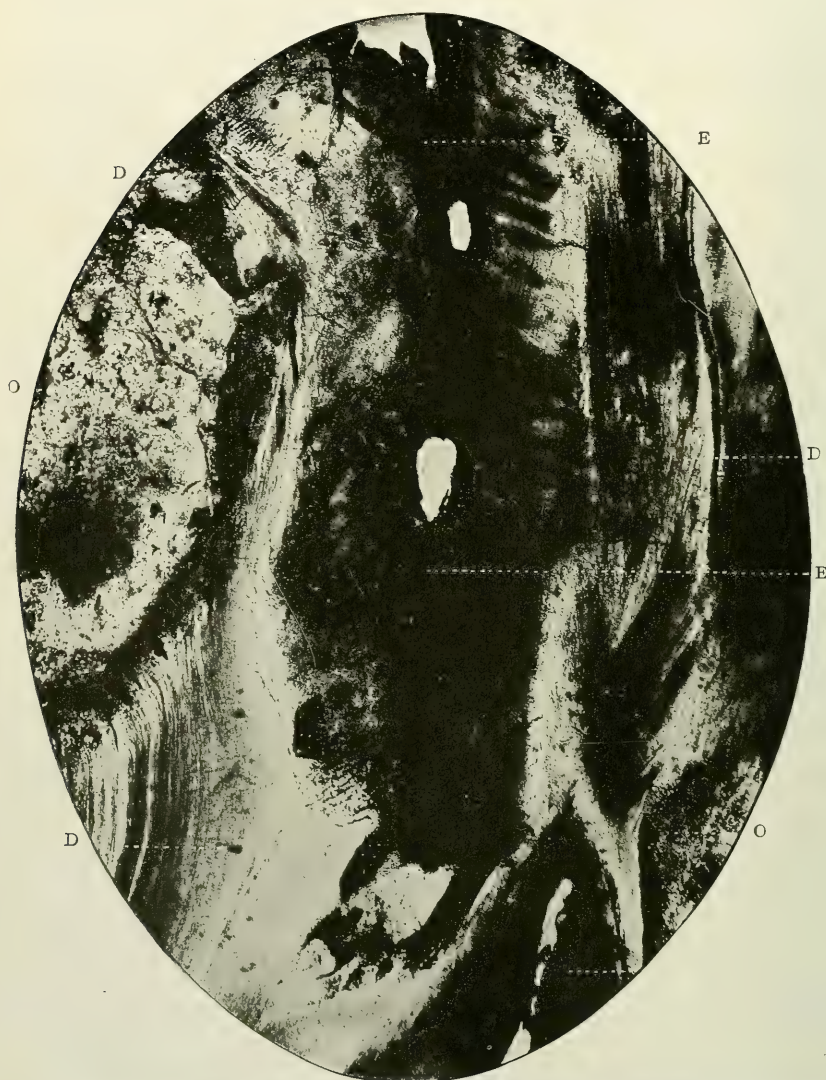
FIG. 343.—Sagittal section of a radicular odontome. Prepared by grind
ing. Unstained. Magnified 50 times. E. Enamel; D. Dentine; O. Osseous
tissue.

fine point of enamel has blended with the intervening mass. Passing
still along the periphery of the neoplasm, and situated at its inner-

most portion, are several radiatory dentinal systems, but no cementum. At its base a slight amount of hyperplasic, lacunated cementum is found. As it passes up to the labial surface once more, the cementum is a structureless, narrow band, approximating accurately to the normal type.

The greater bulk of the tumour consists of fine-tubed dentine, and amorphous osseous substance with occasional large cavernous spaces. Deep down at the base of the second denticle are apparently the remains of three more irregular, dwarfed, rudimentary outlines of incisor teeth, each having enormously thick coatings of deeply

FIG. 344.—Radicular odontome. Magnified 45 times. (*From a section in the collection of G. W. Watson.*)

pigmented enamel of a low type, and fine-tubed dentine. Marked lines of demarcation exist between these dentinal systems and the bony deposits. No signs of absorption can be detected; but in places a few layers of interglobular spaces, and also enormous fusiform cavities, filled with *débris*, occupy the intervening zone.

The odontome is instructive because, being a root tumour, there is less than the ordinary amount of cementum. If one may venture to hazard an opinion as to its genesis and further development, it
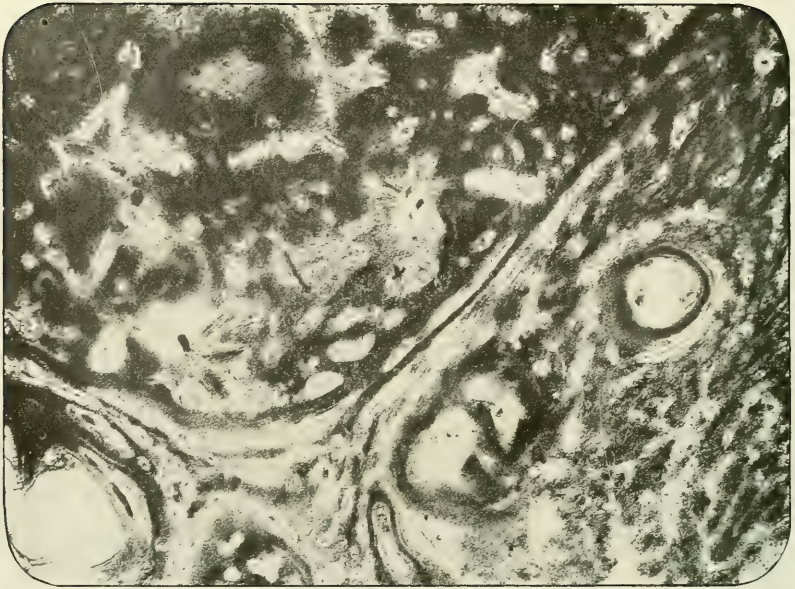
FIG. 345.—Another radicular odontome. Magnified 45 times. (*From a section in the collection of G. W. Watson.*)
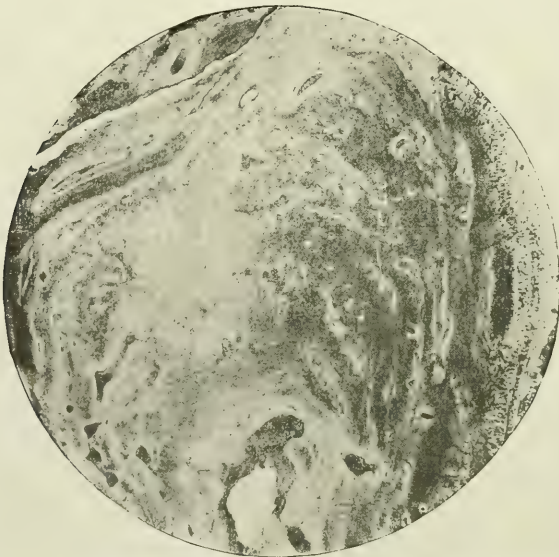
FIG. 346.—Another portion of the preceding. Same magnification.

would seem to have been produced by a fusion of several more or less complete, ill-shaped enamel organs, each of which had attained an unusual degree of physiological activity, which was, however, profoundly modified by certain pathological conditions of growth.

The accompanying illustrations show the histology of two radicular odontomes in the possession of G. W. Watson, of Edinburgh. Fig. 344 exhibits the dentine of the root with a new growth, made up of ill-formed (?) osseous material, of which the following are the chief features:—(i) Large vascular channels; and (ii) matrix containing irregular spaces and branching processes, not, however, sufficiently well organised to be designated lacunæ. In the greater part it is coarsely granular, the matrix being arranged often in more or less spherical bodies of varying sizes. At the periphery there are many large thickened masses of hyperplasic cementum, the enamel and dentinal teeth systems being suppressed.

In Fig. 345 it is seen that the neoplasm bears some resemblance to plici-dentine. Many dentinal systems are present, with very granular dentine matrix and radiating tubes. Cementum and enamel are absent.

Composite Odontomes

Origin.—Aberrations and abnormalities in the development of the whole or part of the constituents of the tooth germ. Apparently they are confined to man, but they are very rare.

Macroscopical Appearances.—An odontome of this type, recorded by Dolamore, appeared in the right molar region of the mandible of a male aged 22 years. Its measurements after removal were 4.2 cm. in length, 2.8 cm. in depth, and 1.6 cm. in width, and total weight 24.5 grammes. This makes it one of the largest ever described.

"*Clinical History.*—The patient stated that he first had pain about three years previous to his visit to the Hospital. He came under the impression that there was a root of a tooth troubling him. There had been swellings which came and went. The suppurative inflammation which occurs almost universally around these odontomes when they 'erupt,' appears to be due to the density of their structure. The soft structures forming the lining membranes of their sockets, even though it is also the forming organ, can have but slight 'hold' upon them and readily become detached. Hence they become more comparable to sequestra than to pulpless teeth,

in which there remains, through the cementum, a gradual transition from the living cells of the pericementum to the dead dentine.

"*Appearance in situ.*—The molar and second premolar teeth are absent on the right side, the first premolar root remaining.   A
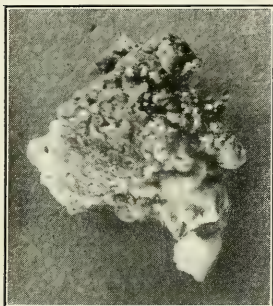
FIG. 347.—A composite odontome from region of a third right mandibular molar.
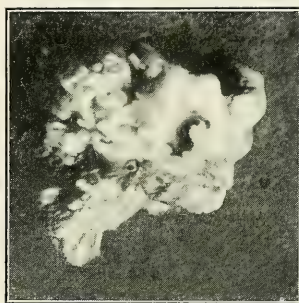
FIG. 348.—Another aspect of same odontome as in preceding figure.

roughened, hard surface occupied about the situation of the second molar tooth, being distinctly posterior to the normal position of the first molar.   A pointed probe could be passed vertically downwards, at the anterior surface of the tumour, to a great depth.   At the

FIG. 349.—Composite odontome, with portions of the capsule adherent to it, and the third molar placed *in situ*.   The tumour is viewed on its lingual aspect.   From an original photograph.   Actual size.   E. Portion which was erupting; W. Crown of third molar.

posterior margin of the exposed surface the probe passed in a slanting direction downwards and backwards.   The margins of the gum were slightly inflamed, and there was a slight amount of pus.   The two surfaces of the mandible were markedly protuberant, the en-

largement extending from the anterior margin of the tumour towards the angle.
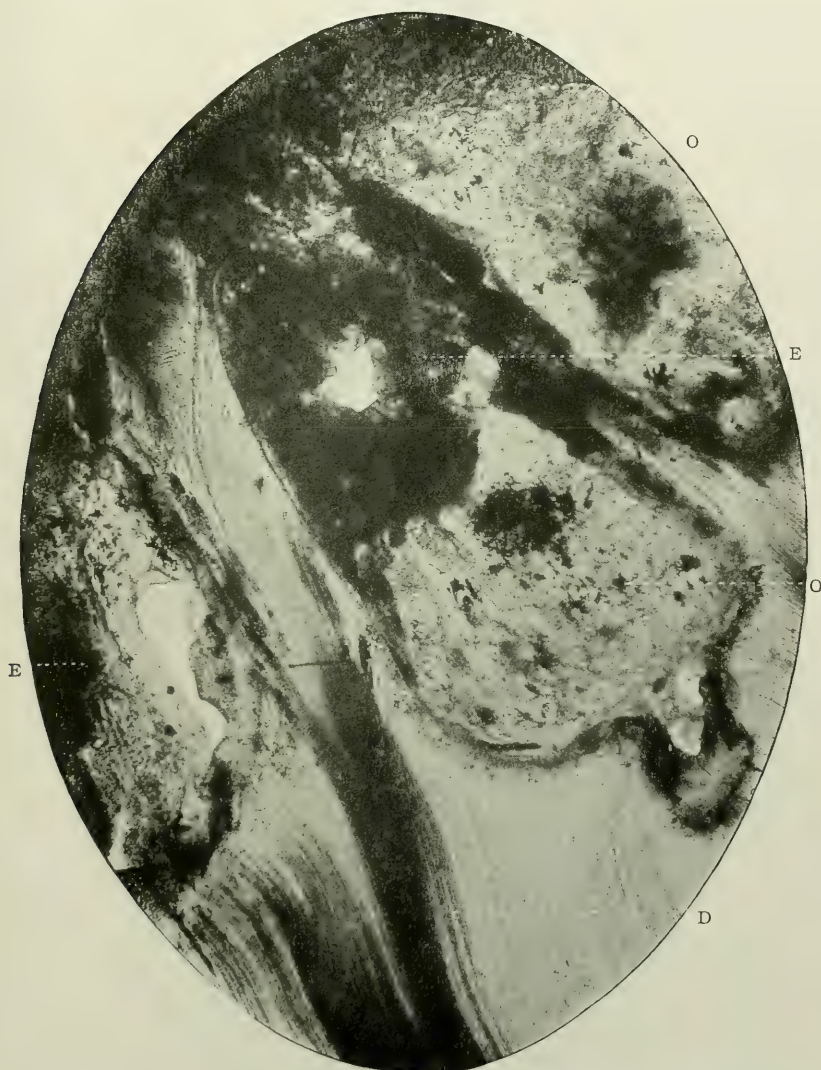
FIG. 350.—A composite odontome. Prepared by grinding. Unstained. Magnified 50 times. E. Enamel; D. Dentine; O. Osseous material.

"*Removal.*—A curved root elevator, passed down the anterior margin, slowly levered the mass out of its bed, gouging out with it,

and adherent to it, a portion of a fibrous-like capsule. On examination, this capsule and the overlying odontome showed on the lower and outer posterior portion the imprint of a molar tooth. This was found implanted in the wall of the 'socket' of the odontome, whence it was readily removed with an upper root forceps. The remainder of the capsule gave to the 'socket' the 'velvety-like lining' mentioned by Jordan Lloyd, in a case he has described.

The size and weight of the odontome, and the comparative ease with which the tumour could be removed with the elevator were outstanding features of the case."

HISTOLOGY

The periphery of this composite odontome is made up of fine-tubed dentine, the constituents of which are arranged in a curiously centripetal fashion. Thus, instead of running from within out, as in an ordinary manner, these pass from without inwards, sometimes for a considerable distance. The centre of the growth contains many small, irregular islands of enamel. Traces of absorption can be seen, where dark bands of enamel have been deposited in the spaces in the dentine matrix. Interglobular spaces very frequently abound.

The "osseous" material (Fig. 354) which composes the great part of the growth is probably merely dentine matrix full of irregular, interglobular spaces and canals. No cementum as such exists; repeated examinations of the section made transversely to the body of the odontome have failed to show lacunæ and canaliculi. In one place, the appearances represented by a crude form of plicidentine may be noted.

An interesting and valuable feature of this tumour in the presence of its fibrous capsule (Fig. 355). Sections of this reveal several structures. First, the soft investing tissues are made up of small round cells with one, and sometimes two, large round nuclei in their interiors, also long fusiform branching cells containing elongated flattened nuclei, all embedded in a delicate white connective tissue stroma. Blood-vessels abound, and are more numerously distributed at the surface, directed towards the body of the tumour.

Sections have been cut in which the relationship of the bone and capsule have not been disturbed. The result shows the probable method of formation of the dentinal substance (Fig. 356). At the attached margin of the capsule, multitudes of osteoblasts cohere to
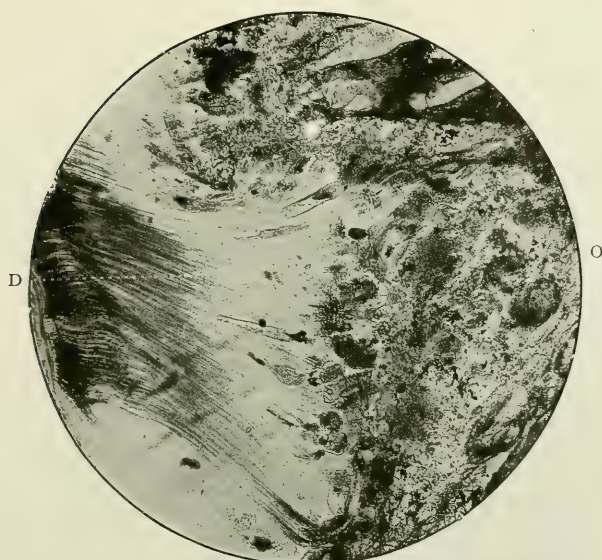
FIG. 351.—Composite odontome. The section is a portion of the periphery of the growth, the dentinal tubes running outwards to the left, the remainder consisting of amorphous dentine matrix with absorption areas intervening. Prepared as in Fig. 343. Unstained. Magnified 45 times. D. Dentine; O. Osseous material.

FIG. 352.—Same as the preceding figure, but from another portion of the periphery. Prepared as in Fig. 343. Unstained. Magnified 45 times.

the margin of the dentogenetic zone, or what corresponds to this zone of formed but uncalcified tissue in developing dentine. This is a clear, translucent band, in which are found not only the familiar calcospherite spherules, but also rudimentary empty tubes (Fig. 357). The osteoblasts are closely placed side by side, and are seen, here and there, to be embedded in cloudlike masses of amorphous stained albuminous material. Osteoclasts sometimes mingle with osteoblasts.

Fig. 353.—Composite odontome. Shows enamel and amorphous dentine fused together, the dark masses being the highly pigmented enamel, the lighter portions the dentine. Magnified 45 times. E. Enamel; D. Dentine.

Here there are apparently direct and irrefutable evidences and proofs that round cells, osteoblastic in shape and nature, are the factors concerned in the building up of dentine, the empty tubes being probably the remains of the connective-tissue stroma of the capsule. Thus, again, are corroborated, in an instructive and un-looked-for manner, the hypotheses which would relegate to the odontoblasts of the pulp a more important function than dentine building, and assign this physiological process to the round osteo-blastic cells seen on the surface of the pulp, as well as on the free edge of the fibrous capsule of this composite odontome.
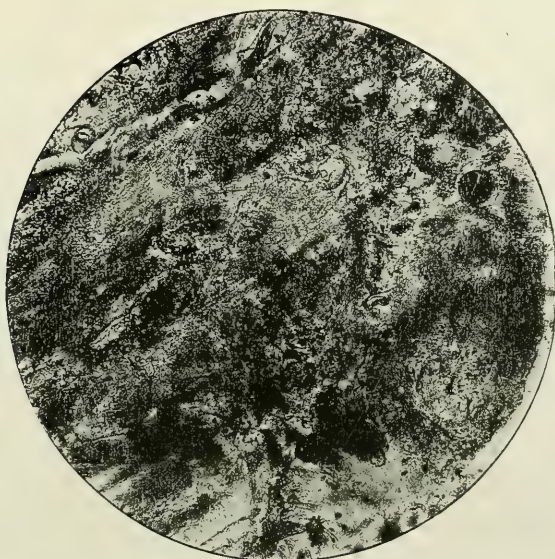
FIG. 354.—To show the amorphous, irregular character of the dentine comprising the greater part of the body of the odontome. Magnified 45 times.
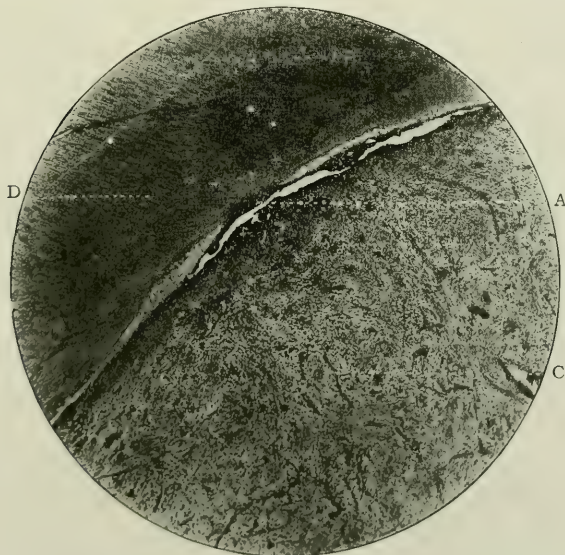
FIG. 355.—Capsule *in situ*. The upper part of the photograph is dentine, the lower the fibrous tissue. Magnified 45 times. D. Dentine; C. Capsule; A, Albuminoid material.
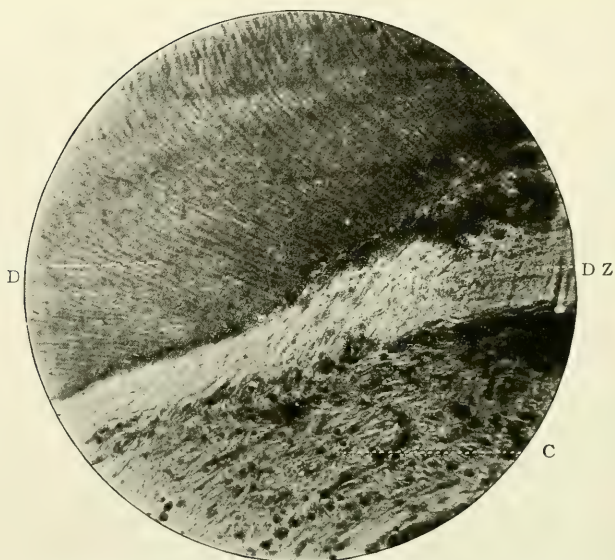
FIG. 356.—Same as the preceding, showing, D. Dentine, C. Capsule, and DZ. Dentogenetic zone intervening. Magnified 240 times.
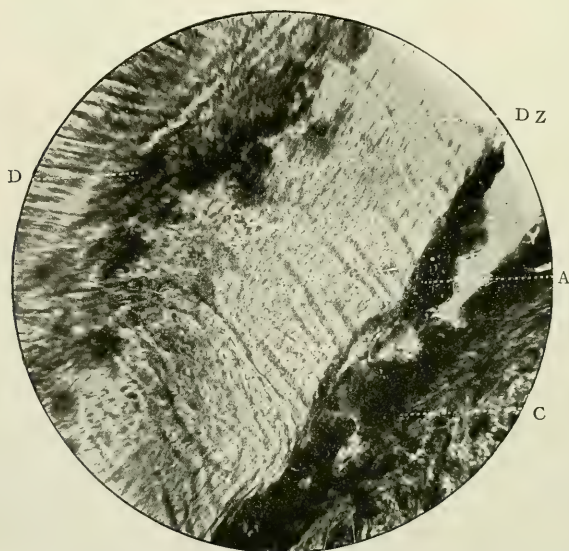
FIG. 357.—The same as the preceding, to show the calcospherite spherules and empty tubes in the dentogenetic zone; below which are dark masses (A) of deeply stained albuminous material. Magnified 600 times.

ODONTOCELES

Similar clinically to odontomes, but differing very considerably in a pathological and pathogenetic sense are the odontoceles or *tooth-cysts*. An odontocele differs from an odontome, which is a *tooth-tumour*, in the facts that it is dissimilar in origin, and that the tooth associated with it shows certain structural deviations from the normal, in which loss or addition of tissue, either from maldevelopment or as a result of inflammatory action, is a conspicuous feature.

Varieties.—(i) Sub-capsular, (ii) Extra-capsular.

INTRODUCTORY

The types which are about to be narrated, possessing a few common characteristics or relationships, are very dissimilar from a pathogenetic point of view, and, at the same time, are extremely remarkable and interesting. They serve to illustrate the belief that greater precision is required of dental surgeons when describing certain pathological changes that may take place in the osseous framework of the maxillæ and mandible.

There is room for an amplification and revision of the nomenclature employed in the subject of dental pathology, and new terms must necessarily be introduced to more accurately represent conditions as knowledge of the special pathology of the teeth advances. It is necessary to throw some light on the obscurity of the origin of cysts of the jaws, and to ascertain in what circumstances or through what agencies fluid is produced in such amount as to constitute the chief clinical characteristic. Their signs and symptoms are unimportant and uninstructive, but the morbid conditions and the patho-histology of the tissues are of extraordinary significance. If the deductions arrived at are inconclusive, or open to another construction, allowance must be made for the difficulties attaching to the research and to the great mystery surrounding the unique display of morbid phenomena.

It will be found that odontoceles can be divided into two classes. Thus one would be designated an example of a *Sub-capsular odontocele*, and the other of an *Extra-capsular odontocele*. They merely fall into the same main category because, in the opinion of the author, they cannot strictly be placed under any other classification.

A common feature here is the existence of a unilocular cyst in the jaws, containing an anomalous tooth—non-erupted, and so-called

"encysted," disclosing itself in adult life. It is incorrect to describe "a case of an encysted tooth;" the fact that it is imbedded in the bone does not of necessity mean a disease of the tooth or jaw, but a state of being or condition in which the tooth happens to exist.

One is led to understand, from the pages of text-books and current literature, that a buried, "imprisoned" tooth, as it is somewhat fancifully called by Roswell Park in *The Principles and Practice of Modern Surgery*, 1908, may induce a passive or active reaction to the containing tissues. Thus (1) it may, usually, remain in its uncommon irregular position undisturbed, unnoticed, inert, impacted or otherwise throughout the life cycle of the individual, giving rise to no signs or symptoms of anomaly; or (2), it may, rarely, originate a tumour, through the production and accumulation of a fluid body around it. Clinically, when a tense, fluctuating, non-inflammatory, painless swelling of the jaw presents itself for diagnosis, either a dental cyst or a follicular odontome is first brought to mind. These two, and especially the former, are the commonest cysts with which the dental surgeon has to deal. But other species of cysts may occur at times, and it is possible to fully enumerate them in their probable order of frequency.

SPECIES OF CYSTS OF THE JAWS, BASED ON THEIR PATHOLOGICAL VARIATIONS

(1) Dental cysts.
(2) Eruption cysts.
(3) Follicular odontomes—simple or compound.
(4) Epithelial odontomes or multilocular cystic tumours.
(5) Mucous cysts of the antrum.
(6) Odontoceles: (*a*) Sub-capsular, (*b*) Extra-capsular.
(7) Cystic adenomata of the antrum and gum.

Of the varieties of odontoceles, the sub-capsular form occurs in the young—from 10 to 20, the extra-capsular form later in life. Two illustrative cases follow.

A

A Sub-capsular Odontocele. (*The case of Mr. R. L.*)

On examination of the mouth a large, smooth, bluish, fluctuating, painless distension of the bone was seen occupying the right canine region of the mandible. Its presence had been detected subjec-

CLASSIFICATION OF CYSTS OF THE JAWS

1. Common characteristics: Non-inflammatory, benign, painless, slowly growing, tense, fluctuating swellings, possessing epithelial linings, and having, except No. B, a tendency to increase in their dimensions, and in the direction of least resistance.

2. Specific characteristics:

Variety	(i) Age	(ii) Origin	(iii) Location	(iv) Cavity	(v) Fluid	(vi) Radiograph
A. Dental.	Adult.	Epithelial "rests" of periodontal membrane.	Apical region of "dead" tooth.	Small, unilocular.	Viscid, yellow, limpid, cholesterine.	Well defined outline.
B. Eruption	Child.	External epithelium of enamel organ and remains of tooth-band.	Between enamel and dental capsule, in gum, over erupting tooth.	Very small, unilocular.	Serous, whitish, transparent.	Nil.
C. Follicular odontome: A. Simple, B. Compound.		A. Stellate reticulum of enamel organ. B. Whole of several tooth-germs.	A. Around unerupted tooth, e.g., premolar. B. Around unerupted molar.	A. Large, unilocular. B. Enormous, unilocular.	A. Large amount, yellow, slimy, cholesterine. B. As above, many denticles.	A. Tooth in cavity. B. Numerous denticles in cavity.
D. Epithelial odontome.	Young adult.	Unatrophied portions of tooth-band.	Region of third mandibular molar.	Large, multilocular.	Thick, caseous.	Tooth may exist in cavity.
E. Mucous cyst of antrum.	Any.	Retention of mucus through obstruction of duct of gland.	Antrum.	Unilocular, polypoid.	Viscid, yellowish.	Nil.
F. Odontocele: A. Sub-capsular. B. Extra-capsular.	A. 10 to 20 years. B. 10 to 40 years.	A. Internal epithelium of enamel organ. B. Tissues outside enamel organ.	A. Mandible. B. Maxilla.	A. Large, unilocular. B. Large unilocular.	A. Thin, watery, yellow, or (if old) blue, clear, abundant. B. As above.	A. Tooth in cavity presents incompletely developed crown. B. Tooth in cavity, presents calcified mass on surface of enamel.
G. Cystic adenoma of antrum.	Adult.	Glandular epithelium of mucosa.	Antrum.	Large, may fill antrum.	Thick, creamy or caseous, yellow.	Nil.

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tively only five weeks previously. The canine was missing, and there was no history of its having been extracted, though several teeth had been removed and a denture was being worn. The first premolar was almost in contact with the distal surface of the second incisor. The corresponding tooth was not in evidence, but both left canines were erupted in correct alignment with the dental arches. A cyst was diagnosed. Radiographs disclosed the following condition of the parts: At the base of an extensive cavity in the bone, measuring 2.5 cm. by 2 cm. by 1.5 cm., was a canine, non-erupted, non-impacted, non-absorbed. It was placed vertically in the jaw, its crown pointing upwards and extending into a some-what triangular hollow, produced by the divergency of the roots of the second incisor and first premolar. Examination of these roots showed no visible areas of absorption whatever, though the latter appeared to be twisted. On inspecting the radiograph of the canine it was at once apparent that there was a loss of substance—*i.e.*, enamel and dentine—at the summit of the cusp. It was possible to trace a direct line of continuity between the cystic contents out-side the tooth and the pulp itself.

On excising the cyst wall there was no venous hæmorrhage, as had been anticipated from the colour of the tumour. Instead, the cystic contents were deeply discoloured and almost black. It was this that had imparted the blue appearance by reflected light to the oral tissues, a phenomenon similar to that frequently witnessed in connection with hydroceles of the *tunica vaginalis*. The tooth was removed and the cyst wall dissected out; granulation tissue soon formed, and at the end of ten months healing had taken place, a mere shallow depression on the surface of the jaw marking the site of the odontocele.

The tooth was at once carefully tested for the presence or absence of Nasmyth's membrane. The inner layer was found, but no cellular layer. It was then treated by the Koch-Weil method, and the section showed that the tip of the crown was defective and its growth had never been completed. There was no absorption of tissue. A hollow, tube-shaped core occupied the vertical axis of the upper part of the tooth. Had it erupted in the ordinary manner it is impossible to guess what the consequences would have been. Soft tissue, composed of small cells, extended vertically into the pulp cavity, and also slightly laterally, in places, into the dentine. The pulp itself was composed, at its coronal portion, of broken-down cells, the odontoblasts were unrecognizable, the nerve bundles

obliterated, while a few long endothelial-like cells closely applied
in bundles represented the collapsed and shrunken walls of the
vascular system. It is impossible for anyone to say what had hap-
pened to the enamel organ at that part which was going to form the
summit of the crown. It could not have been due to septic disease
of a deciduous predecessor, for one would then probably get the
whole of the enamel organ deranged with regard to its usual func-
tions. Apparently there was a sudden cessation of the work of the
ameloblasts at that spot, leaving not only a breach of surface but a
direct opening into the pulp cavity. After suitable preparation the
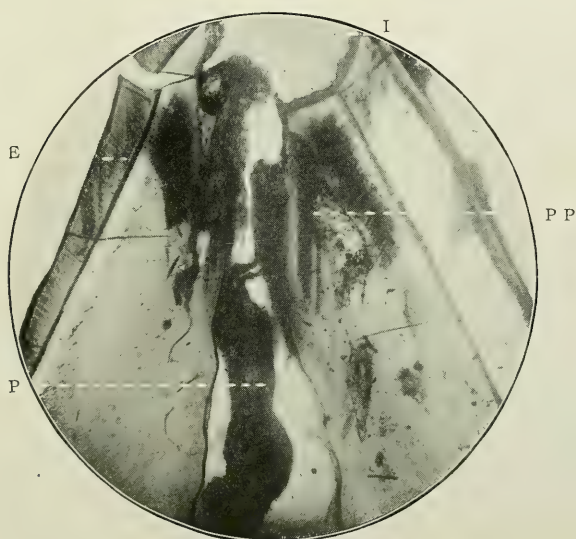
FIG. 358.—Longitudinal section of canine described in text. E. Enamel;
P. Pulp cavity; PP. Pulp tissue extending laterally into dentine; I. Incisive edge
of tooth. Magnified 20 times.

cyst wall was microscopically examined. It was very thin, only
measuring 0.25 to 0.75 mm. Composed of firm fibrous tissue it had
a lining of numerous soft, large epithelial cells, held loosely together
by a frail cementing substance. It was in no sense a compound
epithelium, as seen sometimes in both dental cysts and follicular
odontomes, nor were they secretory cells. It was, probably, the
external epithelium of the enamel organ swollen by absorption of
cystic fluid, and the cyst wall was nothing more nor less than the
dental capsule itself.

To the accident of the presence of a congenital lesion of the hard

parts of the crown, and not to impaction or delayed eruption, can be attributed the evolution of this cyst. Here is, then, an example of a sub-capsular odontocele—*i.e.*, a cyst originating beneath the capsule or follicle of an abnormal tooth, as a result of the effusion of lymph from the neighbouring blood-vessels, into a potential cavity produced by a developmental defect of the summit of its crown. It may be suggested that the same morbid phenomena might have been induced by serous exudation into a space or spaces of a vacuolated follicle. But here the capsule had undergone no such retrogressive metamorphosis on account of its being retained in the jaw, as already mentioned. Hence the opinion that a cavity had existed through something unknown happening to the formative cells of the enamel organ many years ago, and effusion of lymph into the cavity thus created.

B

An Extra-capsular Odontocele.[1]   (*The case of Dr. C. N.*)

Interesting and instructive as is the case just detailed, the second variety of odontocele is even more remarkable.

Past history: The maxillary incisors and premolars had previously been removed. One was broken, two were quite sound, and caries was present in the rest; the sound teeth were sacrificed for the purposes of giving firmer hold to a bridge which was to be constructed. The denture was worn for ten years. About three weeks before the tumour "became cystic"—to quote the patient's own words—he broke the plate through the middle, and he continued to wear it, being too busy to have it attended to. He believes that the movements of the fractured appliance irritated the jaw, and "stirred the latent tumour into activity." His dentist pointed out to him ten years previously that the permanent maxillary canine in the right side was missing, and had apparently never erupted.

Present history: "The patient's attention was first directed to a small enlargement of his cheek. He noticed it when washing his face, being conscious that the right cheek was fuller than the left. On feeling it more carefully he could make out a round, hard prominence close by, and at the same level as the right ala of the nose. His first impression was that an alveolar abscess was forming, but

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[1] A similar case—the second to be recorded—was detailed by Mr. J. Alan Forty and the author in a paper presented to the International Dental Congress. 1914.

it did not make any progress in the way of 'ripening.' About two weeks later a little swelling appeared in the mouth in the angle between the upper lip and the gum, and in the region of the second incisor and canine tooth. This was fluctuant, and the patient thought that it probably contained pus. He incised it, but did not evacuate any pus, and in a few days it had attained the same size again. He then called in a brother practitioner, who incised it down to the bone, and it was then apparent that the swelling was a cyst. It soon filled up and became considerably larger, and the patient then thought it advisable to consult a surgeon with the view to its removal. Up to this point there was no pain associated with it beyond a throbbing in the gum and part of the hard palate.''

FIG. 359.   FIG. 360.

FIG. 359.—A right maxillary first incisor contained in an extra-capsular odontocele in a boy of 10 years. Labial aspect. Shows a calcareous nodule attached to the enamel. (*Mr. Alan J. Forty's case.*)

FIG. 360.—Another aspect of tooth of preceding figure. Shows a calcareous nodule attached to the enamel.

"The patient saw a leading operating surgeon who examined the cyst and advised its removal. A few days later he proceeded to do this, and on dissecting off about a third of the cyst wall he found it firmly rooted to the bone of the maxilla. He sheared off the dissected portion of the cyst close to the bone with the view to its pathological examination and further operative treatment. From the clinical appearance he was of the opinion that the tissue was malignant in character. Examination was made of the detached portion, and on the patient's return two days later, he was informed that the tumour was of the nature of a myeloid sarcoma. This diagnosis, however, was disputed by the pathologist who, as a personal friend of the patient, was called in to give an opinion. He was very emphatic that there were no tissue elements to warrant a diagnosis of malignant disease, and it is only fair to the operating surgeon to say that he abandoned the diagnosis of myeloid sarcoma, and consented to perform a much more modified operation than he originally intended, although still feeling that there was a malignant

element in the case. The operation decided upon was to remove the affected area with a margin of healthy tissue around it. This was done, a wedge-shaped portion of the upper jaw being removed, and a considerable part of the bone of the anterior wall of the antrum included, the membrane being left. The cavity in the mouth healed up without any difficulty, although the antrum became affected and necessitated douching through the nose. Eventually the wound closed completely, and the mouth has remained sound and well after a period of twelve months." (Patient's report.)

Appearance of the tumour: The specimen consisted of a V-shaped piece of tissue bounded internally by the median line of the palate, and externally by a line running backwards and inwards through
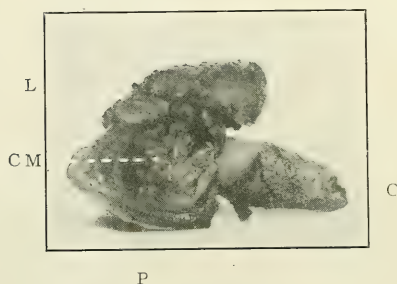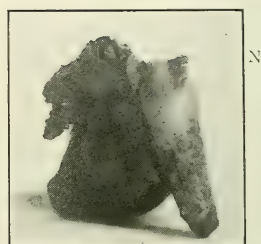
FIG. 361. FIG. 362.

FIG. 361.—Extra-capsular odontocele, as viewed from *mesial* aspect; P. Palatal surface; L. Labial surface, where fluid accumulated; C. Canine; C.M. Calcified mass.

FIG. 362.—Tooth everted from its surroundings to show calcified nodule (N) attached to surface of crown.

the premolar region. Its width in front was 3.5 cm. The labial aspect showed an opening in the alveolar process 22.5 mm. long, 9 mm. wide, the lower margin being placed 6 to 9 mm. from the free edge of the alveolar process. It was made by the incision for the evacuation of the cystic fluid. At its base, and at a distance of about 4 mm. from the surface, lay a calcified mass. The main object in the preparation was a canine fully formed and well developed, 25 mm. in length. Its crown was almost entirely embedded in a solid mass of brittle, calcified material, which was yellowish in colour and partly translucent by reflected light. During the digital disturbances consequent on the operation this calcified cap had obviously been dislodged from the position it had occupied for many years and had become detached, and the crown of the tooth was shelled out from it with the exception of a small excrescence,

which, still adhering to its distal aspect, gave the clue to the patho-
logical conditions which had been occurring. The nodule measured
4 mm. in length, the dimensions of the hard cap being 14 mm. by
12 mm. There were no marks of absorption on the surface of the
enamel. Nasmyth's membrane was present. The calcified mass
was obviously the dental capsule, which had undergone a retro-
gressive metamorphosis. It had not been completely infiltrated,
as portions still retained their fibrous characters (Fig. 361). Patho-
logical calcification or petrifaction occurs almost without exception
only in degenerating, dying, or dead tissue as Hektoen and Riesman
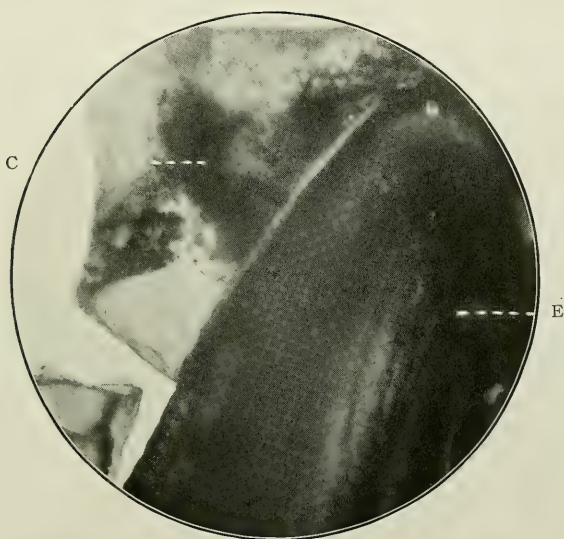
FIG. 363.—Photomicrograph of crown of tooth described in text. E. Enamel;
C. Calcified nodule. Magnified 45 times.

record (*op. cit.*). Thus its appearance can probably be explained
by the fact that the dental capsule of the unerupted canine had died,
and formed a nidus for the lime-salt infiltration, thus differing very
greatly from the follicle in the preceding Sub-capsular odontocele.
The composition of the hard mass was found to be calcium car-
bonate largely. On placing a fragment under the microscope,
and allowing a weak solution of hydrochloric acid to run, by capillary
attraction, beneath the cover glass, bubbles of gas were immediately
evolved, the residue of organic material presenting an amorphous
structure with a tendency to the formation of a pattern of spherules.
Around the surface of this calcified capsule was developed the cyst,

and what was apparently the "bone of the jaw" mentioned in the patient's notes was its exposed surface. Histologically examined, the soft parts consisted chiefly of connective-tissue fibres with nmuerous small cells, changed connective-tissue corpuscles, tiny hæmorrhages, clusters of fat cells, blood-vessels, and a small amount of bone which represented all the remains of the thinned and expanded external alveolar plate. The cyst wall was lined with epithelium. There were no microscopic signs of inflammation.

Whence came the cystic fluid? It is difficult to determine. Eliminating every possible structural element in the gum which could give rise to a cyst, it might be conjectured that the cells of the walls of certain lymph spaces in the submucous tissues had broken down, and produced, by rapid multiplication, the cyst, through irritation from the friction of a loose, fractured mechanical appliance on the one side, and the hard unyielding surface of the ossified capsule on the other.

At first sight the dental condition might have been regarded as a calcified follicular odontome. But this was not so. For the cystic fluid was *outside*, not *inside*, the capsule, which was not expanded, but remained, in spite of its petrifaction, closely adherent to the surface of the tooth. Hence it is an extra-capsular odontocele. Its origin may be explained by the breaking down of epithelium or endothelium in the soft tissues of the jaw lying between, and stimulated into growth by the pressure, and perhaps friction, of two hard, unyielding substances.

There are some further problems surrounding this case which can never possibly be solved: The age of the patient when calcification of the capsule took place; the possibility or otherwise that this calcification, acting as an obstruction, was the cause of the non-eruption of the tooth; the histological characteristics of the actual structures in the soft superimposed parts, which, on breaking down, produced the odontocele; the date of its formation; and the nature of the still more mysterious forces which governed the impregnation with lime salts of the dead or dying follicle.

The fact that mechanical agencies play a not inconsiderable part in the production of disease calls for no special comment. And teeth which undergo moliminous eruption, or do not erupt at all, as is so frequently the case with the third molars, may act as foreign bodies and produce inflammation and absorption of the surrounding parts, and even give rise to cystic tumours of the maxilla.

CHAPTER XVI

ORAL MICROBIOLOGY

Introduction—Classification of plants—Classification of *Bacteria* and
   Protozoa—Microscopic examination of oral material—Pathogenic
   organisms—The Pyogenic cocci—Filterable viruses—Adventitious
   bacteria of the mouth—Micro-organisms of dental caries.

INTRODUCTION

In contributing a chapter on Oral Microbiology, one can but briefly
outline the classification and characteristics of the organisms com-
monly found in the oral cavity. It would be inexcusable to attempt
the presentation of more than the dental phase of the subject, there-
fore the reader is referred to standard works on the subject of Micro-
biology, Bacteriology, and Protozoology for a detailed account of
the micro-organisms.

The oral cavity is a fertile field for the development of various
types of micro-organisms. We find there a temperature suitable for
the growth of a large percentage of the organisms, substances
supplying nourishment, a sufficient supply of moisture and oxygen.
Furthermore there are various crypts and spaces in which they may
develop undisturbed. The organisms manifestly gain access by
way of the mouth, being carried there with food, drink, instruments,
etc., or, they may be carried in through the nasal passages with the
inspired air. These organisms may be transmitted directly from
other individuals. The investigator possessing this knowledge is
not surprised at the variety found, but rather that more species
are not encountered.

Some micro-organisms do not find a suitable environment, and they
will not multiply at all, or but to a very slight extent, in the oral
cavity. Some are antagonistic to the development of others.
Foods, drink and the saliva are constantly or intermittently not only
diluting the organisms, but carrying many of them along the pas-
sages into the gastro-intestinal tract, causing a diminution of the
numbers and also changing, to some extent, the types found in a
given individual from day to day.

Many of the organisms present are of no pathological importance, being of a type incapable of exerting a detrimental action on the tissues. Others, while possessing no pathogenesis in the strict sense of the word, are of great importance to the dental practitioner and pathologist owing to the fact that they play an important part, either directly or indirectly, in the condition known as dental caries. Various types of organisms are also encountered, which are capable of producing definite infections when local and general conditions such as will admit of their invading the tissues exist.

On studying microscopically and culturally, the flora and fauna of the oral cavities of a large number of individuals, the investigator is always impressed with a number of facts of interest and also of great import. The most important of these are as follows:

(1) The number of organisms in the oral cavities of different individuals varies markedly.

(2) The more closely associated are individuals, the more nearly, other things being equal, does the micro-organismal content of their oral cavities correspond.

(3) The number and types of micro-organisms vary in the oral cavities of all individuals to a great extent according to environmental conditions, food, etc.

(4) The micro-organismal content is influenced directly by the degree of cleanliness the individual exercises as regards his oral cavity.

(5) However cleanly one may be, it is impossible to remove or prevent the growth of all micro-organisms.

(6) Certain types of organisms are encountered almost universally in different individuals.

(7) Micro-organisms possessing a definite pathogenicity may exist in the oral cavity of an individual without causing any untoward results.

The greater number of the micro-organisms encountered in the oral cavity are Bacteria. Higher Fungi are found, a few varieties of Yeasts, and several varieties of organisms of various types, ordinarily known as Moulds. Some of the latter are capable of producing pathogenic conditions, which, however, are not common. The protozoa have not been studied in the same detail as bacteria. There are, however, comparatively few varieties present.

Before entering into a discussion of the various types of organisms found in the oral cavity and their significance, it is essential that the

student be possessed with some knowledge of their morphology and physiology, also of their place in the biological world.

Bacteria are microscopic, unicellular vegetable organisms, occurring either as small spheres, straight or curved rods, or as long threads, motile or non-motile, some forming endospores, multiplying by a process of transverse division or fission, devoid of chlorophyll, and nourished by a process of osmosis.

These organisms are very minute and the individual cells can only be seen when magnified several hundred diameters; they average in size from about 0.4 to 1.5 μ in diameter for the spherical forms, and from 0.8 to 4 μ long for the rod shaped, and 15 to 30 μ in length for the thread forms.

Each individual organism consists of a single cell: in no instances are bacteria multicellular. The majority of the organisms classified as bacteria are vegetable, and therefore belong to the plant kingdom. The morphological classification basically depends on the shape of the individual cells, as will be seen by consulting the list given, page 413. They are devoid of chlorophyll, the green colouring matter of many plants, which enables them to obtain nourishment, at least partially, from inorganic sources; consequently they are nourished by a process of osmosis. They divide by a process of transverse division or fission: therefore they are classed with the Schizophyta, or fission plants under the name of Schizomycetes or fission fungi, belonging to the group of plants known as Cryptogams.

Certain of the bacteria possess long slender appendages, which can only be demonstrated by special methods of staining, called flagella. These are constantly in motion, and cause the organisms to move about in the substratum in which they are developing. The rapidity and mode of the movements give to the organisms a characteristic type of motility; many non-flagellated bacteria present an oscillatory motion known as Brownian or Brunonian motion.

Certain bacteria produce small oval or spherical, highly refractile, glistening bodies, known as *spores or endospores*. When certain conditions are present, as deficient nutritive material, or moisture, or the presence of substances exerting a detrimental action on the organism, small granules (the sporogenic granules) appear within the cellular protoplasm; these granules gradually coalesce in the centre or at one end of a cell forming the endospore. These endospores lie dormant until conditions are favorable for their development, when they germinate and pass into the vegetative stage. The vegetative

cell is capable of multiplying by a process of transverse division. The statement is frequently encountered that some bacteria reproduce by sporulation; this statement is, according to our present views, erroneous.

In cultures of certain bacteria, cells are found which vary markedly from the normal types in size and shape. In some instances these cells have been called *Arthrospores*, the claim being made that they play a part in the reproduction of the organisms. These cells are in all probability involution forms of the organism, due to osmotic disturbances, and not true arthrospores.

Yeasts are small oval or spherical vegetable organisms which reproduce by a process of budding, at times producing small spherical or oval bodies known as ascospores. They are classed under the Eumycetes with the group of Ascomycetes.

The *Moulds* include a group of organisms of various types whose common characteristics lie in the fact that they develop from spores forming a mycelium, which is the body of the fungus, to which are attached hyphæ of various types, at the ends of which outgrowths appear which carry the spores or conidia, endospores or gonidia; or bodies are formed by the union of elements at the end of two hyphæ, joining together, known as zygospores.

There is a group of organisms allied to the yeasts and moulds which have resisted all attempts at proper classification, and are therefore grouped under the head of *Fungi Imperfecti*. Many members of this group are of great import from a pathological point of view. Some are encountered in the oral cavity.

The following will give the student an insight into the relationships existing between the various plant groups.

CLASSIFICATION OF PLANTS

(A) *Spermatogams*, Spermatophytes, or Phanerogams, the seed plants. In this group are included the higher plants; all those reproducing by dissemination of seeds.

(B) *Cryptogams*, including those plants reproducing by means of endospores or gonidia, exospores or conidia, budding and by transverse division of fission.

Classification of *Cryptogams* with special reference to those organisms of importance from a pathological viewpoint.

Division I. *Myxothallophyta*, the slime fungi.

Division II. *Euthallophyta*, the true thallophytes, namely those plants which cannot be differentiated into root, stem and leaf.

(A) *Schizophyta*, fission plants:—
  a. *Schizomycetes*, fission fungi, cleft fungi, or bacteria,
  b. *Schizophyceæ*, *cyanophyceæ* or blue green algæ.

(B) *Phycomycetes*, fungi resembling algæ:—
  a. *Oomycetes*, aquatic, not important,
  b. *Zygomycetes*, including many of the important moulds producing zygospores.

(C) *Eumycetes*, true fungi:—
  a. *Ascomycetes*, important as certain pathogenic yeasts and allied organisms are included in this group,
  b. *Basidiomycetes*,
  c. *Fungi imperfecti*, important. This group includes a number of types, some pathogenic, the characteristics of which are of such a nature as to render it difficult to properly classify them.

Division III. *Thallophyta*, including green, brown, and red algæ, not important.

Division IV. *Bryophyta*, including liverworts and mosses, not important.

Division V. *Pteridophyta*, including the ferns and other plants as lycopodium and adder's tongue, not important.

CLASSIFICATION OF BACTERIA

Order: *Schizomycetes*

A. Sub-order: *Eubacteriaceæ* (without coloured granules in cell contents; uncoloured except in a few species, which may produce a soluble or an insoluble pigment).

  I. Family: *Coccaceæ* (globular, becoming slightly elongated before cell division; cell division in one, two or three directions).

  (*a*) Genus: *Streptococcus* (cell division in one direction, united in chains, non-flagellated).

  (*b*) Genus: *Micrococcus* (cell division in one, two or three directions with separation of the cells, non-flagellated).

  (*c*) Genus: *Sarcina* (cell division in three directions, united in packets of eight, non-flagellated).

  (*d*) Genus: *Planococcus* (cell division in one, two or three directions, cells separate; flagellated).

(*e*) Genus: *Plano sarcina* (cell division in one, two or three directions, cells united in packets of eight; flagellated).

II. Family: *Bacteriaceæ* (cells straight, cylindrical, short or long rods without a sheath, non-branching; flagella present or absent; endospores present or absent).

(*a*) Genus: *Bacterium* (cells straight, cylindrical, short or long rods, non-motile; flagella absent; endospores present or absent).

(*b*) Genus: *Bacillus* (cells straight, cylindrical, short or long rods, motile, with peritrichous flagella varying in number, endospores present or absent).

(*c*) Genus: *Pseudomonas* (cells straight, cylindrical, short or long rods, occasionally in short filaments; motile; with flagella arranged at one end, monotrichous or lophotrichous; endospores known in but a few species).

III. Family: *Spirillaceæ* (cells more or less curved; rigid or flexile, cell division transverse to long axis of cell; flagella present or absent, monotrichous or lophotrichous).

(*a*) Genus: *Spirosoma* (cells rigid, non-motile, without flagella).

(*b*) Genus: *Microspira* (cells rigid, motile, with one or two polar flagella).

(*c*) Genus: *Spirillum* (cells rigid, motile, possess a tuft of polar flagella).

(*d*) Genus: *Spirochæta* (cells flexile, sinuous, corkscrew-like, do not possess flagella but are motile, usually actively; motility due to undulations of membrane). (The majority, if not all, of the Spirochæta divide by [a different process than the bacteria and should [be classed with the Protozoa under the Flagellata. Genus: *Treponema*).

IV. Family: *Mycobacteriaceæ* (cells straight, short or long, cylindric, clavate, cuneate in form, at times showing a true branching, or as long, branched mycelial filaments; no sheath; without endospores, but at times forming gonidia-like bodies due to transverse segmentation of cells).

(*a*) Genus: *Mycobacterium* (cells commonly short, cylindrical rods, sometimes bent and irregularly swollen, clavate or cuneate; may present Y-shaped forms or longer filaments with true branchings; produce short coccoid elements which may be gonidia).

(b) Genus: *Streptothrix* (cells commonly long branched filaments; produce gonidia-like bodies; form aerial hyphæ in cultures; causing resemblance to moulds).

V. Family: *Chlamydobacteriaceæ* (cells in the form of filaments, and surrounded by a distinct sheath; cell division transverse or in three directions, resulting in formation of gonidia-like bodies which may or may not be motile).

(a) Genus: *Leptothrix* (filaments unbranched; division transverse).

(b) Genus: *Phragmidiothrix* (filaments unbranched; divisions in three directions; sheath scarcely visible).

(c) Genus: *Crenothrix* (filaments unbranched, division in three directions; sheath distinct).

(d) Genus: *Cladothrix* (filaments show false branching).

B. Sub-order: *Thiobacteriaceæ* (cells show presence of coloured granules, or sometimes diffuse colouring red or violet).

I. Family: *Beggiatoaceæ* (filamentous; with or without sheath; motile or non-motile sulphur granules in cell contents; gonidia formation not known).

II. Family: *Rhodobacteriaceæ* (cells irregular, globular, oval, cylindrical, non-filamentous; contents show the presence of sulphur granules or bacterio-purpurin, red or violet).

PROTOZOA

The Protozoa are microscopic, unicellular, animal organisms of various forms. Some occur in the oral cavity, but have not been studied in the same detail as the Bacteria. The most important are the following:

Of the Flagellata:

Genus: *Treponemata*, several species.

Genus: *Trichomonas*, one or more species.

Of the Sarcodina:

Genus: *Endamœba*; endamœba gingivalis of Gros.

Other endamœba?

MICROSCOPIC EXAMINATION OF ORAL MATERIAL

Various representatives of the different groups, in the foregoing classification, will be observed when one mounts and examines microscopically preparations of scrapings from the oral mucous
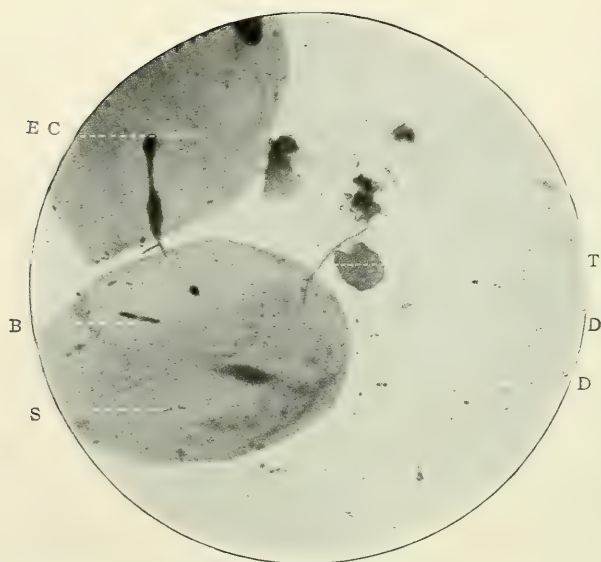
FIG. 364.—The micro-organisms from a healthy mouth direct. Stained by Gram's method. Magnified 800 times. E.C. Squamous epithelial cell from surface of cheek; B. Rod-shaped form, probably *Bacillus buccalis maximus;* T. Thread form; D. Diplococci, probably *Streptococcus brevis;* S. Spiral form.
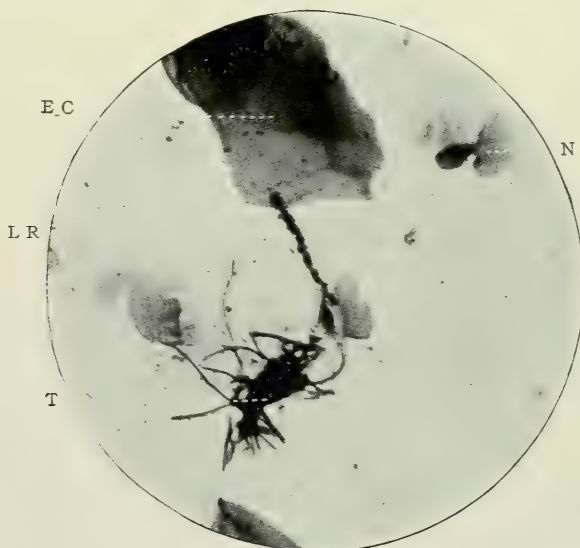
FIG. 365.—From same preparation. Balsam preparation. E.C. Epithelial cell; N. Nucleus of partially-digested epithelial cell; T. Thread forms; L.R. *Leptothrix racemosa.*

membranes, especially in the buccal and alveolar sulci; from the surface of the teeth; collections of food débris on the teeth, or in protected surfaces; soft salivary calculus; exudates from alveolar abscesses, gingival infections and various pathological processes in different parts of the oral cavity.

Suppose a mucinous plaque, from the surface of a tooth, is selected for study, and slide preparations are made, both fresh and stained, for examination. Examination of such stained preparations will reveal the presence of *long thread-like organisms*, some
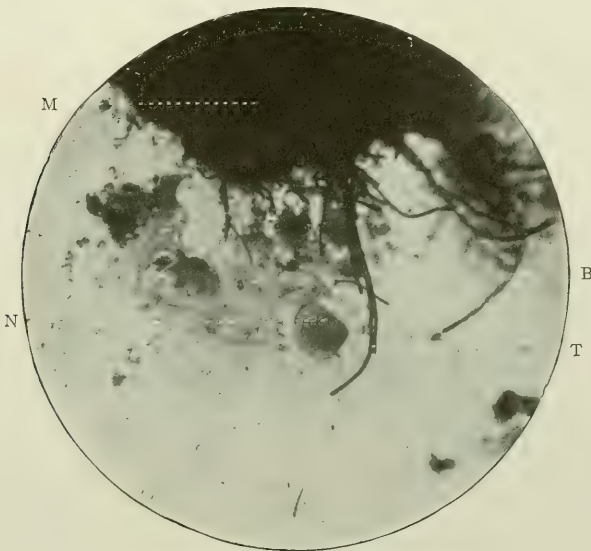
FIG. 366.—From similar preparation to the preceding. Stained by Gram's method. M. Dense mass of micro-organisms from edges of which project: T. Thread forms; and B. Bacillus forms; N. Nucleus of partially-digested epithelial cell.

stained in a homogeneous manner, others presenting small granules. Some of these are species of the genus leptothrix, others the genus cladothrix of the sheathed bacteria, while still others are members of the streptothrix group. Various types of *rod-shaped organisms* may be found, short and long rods, some grouped singly others in chains. Numerous types of *spherical cells* (cocci), some of which present a characteristic grouping, are always found. If fresh, moist material, either slide or hanging-drop preparation, is examined, some of the organisms will be found to possess the power of locomotion and move slowly or actively about the microscopic field.

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Preparations made from the alveolar sulci of unclean mouths, or from pathological conditions may contain bacteria of the same types as described above. In addition there will be found squamous epithelial cells in various stages of disintegration, some containing bacteria; mononuclear and polynuclear leucocytes, frequently present in sufficient numbers to constitute pus; other types of bacteria, as fusiform bacilli, short and long chain streptococci; protozoa and various types of spirochæta or treponemata, which are readily recognised by the fact that they appear as long sinuously curved
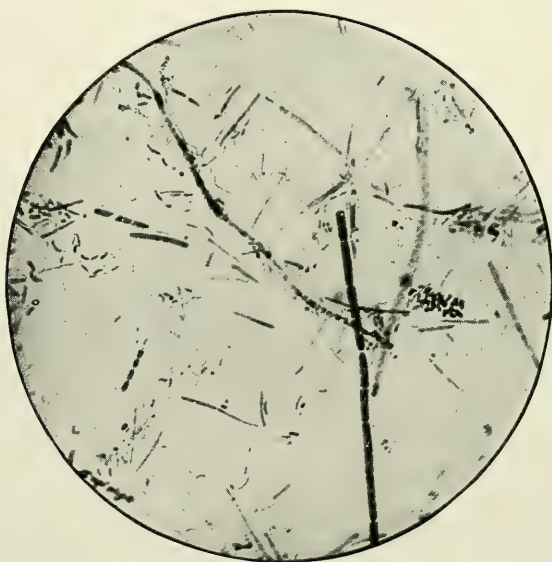
Fig. 367.—The scrapings from an approximal surface of a tooth. Shows all the typical forms found in the mouth. (*Photomicrograph by Leon Williams.*)

rods pointed at the ends, and the *Endamœba gingivalis* recognised as a unicellular organism, many times the size of the largest coccus, circular or irregular in outline containing in the protoplasm granular débris, bacteria, other cells and one or more nucleus-like bodies. Examination of fresh moist preparations of such material will demonstrate the fact that some of the bacteria present are actively motile; the spirochætæ present a rapid sinuous or rotary movement, while the endamœbæ move by means of pseudopods, producing the characteristic amœboid motion.

Preparations examined, microscopically, by aid of one or another

of the dark field illuminators, in which the field is dark and the cells and other particles highly illuminated, will reveal the motile organisms moving about in various ways, some so characteristically as to enable the observer to establish their identity. This method of study will in many instances reveal the presence of organisms not readily demonstrated in stained preparations, notably certain flagellates the significance of which has not as yet been determined.

Many expect a microbiologist to be able to identify and name the various species of micro-organisms, from their microscopic appearance; this is impossible excepting in a few instances. The identification of a species is an entirely different proposition from identifying a family or genus; species closely allied morphologically frequently show material differences when studied individually under appropriate conditions.

For the purpose of identifying species it is absolutely essential, excepting in a few instances, that the organisms be isolated one from another in what is known as a pure culture, *i.e.*, a culture which contains but one strain of a species. Individual organisms are identified by correlating the information obtained by observing the morphological characteristics; the mode of growth on various artificial media; the action on various proteins, carbohydrates, fats and other substances; the relation of growth to various physical conditions, such as oxygen and temperature relationships; pigment production and the determination of whether or not the organism is capable of producing disease.

The methods of separating bacteria in pure cultures from mixtures containing different species, and methods employed in identifying those isolated, are dealt with in works on bacteriological technique; to which the student is referred for such information.

PATHOGENIC ORGANISMS

The following is a list of the most important pathogenic bacteria and other fungi encountered in the oral cavity:

Micrococci:

    Micrococcus aureus. Synonym: Staphylococcus pyogenes aureus.

    Micrococcus albus. Synonym: Staphylococcus pyogenes albus.

    Micrococcus citreus. Synonym: Streptococcus pyogenes citreus.

Diplococci:

    Micrococcus catarrhalis.

    Meningococcus. Synonym: Diplococcus intracellularis.

    Gonococcus. Synonym: Diplococcus gonorrhœæ.

    Pneumococcus. Synonym: Diplococcus pneumoniæ.

Tetracocci:

    Micrococcus tetragenus. Synonym: Sarcina tetragena.

Streptococci:

    Hæmolytic type.

        Streptococcus pyogenes.

        Streptococcus anginosus.

    Non-hæmolytic type.

        Streptococcus viridans: Produces methæmoglobin.

        Streptococcus salivarius.

Bacilli:

    Bacillus diphtheriæ. Synonym: Bacterium diphtheriticum.

    Bacillus pseudo-diphtheriæ. Synonym: Bacterium pseudo-diphtheriticum.

    Bacillus fusiformis. Synonym: Mycobacterium fusiforme.

    Bacillus tuberculosis. Synonym: Bacterium or Mycobacterium tuberculosum.

    Bacillus mallei. Glanders bacillus.

    Bacillus mucosus capsulatus. Synonym: Bacterium mucosum capsulatum.

    Bacillus influenziæ.

    Bacillus anthracis.

    Bacillus aerogenes capsulatus.

    Bacillus tetani.

Streptothriceæ:

    Streptothrix bovis. Synonym: Streptothrix actinomyces, Actinomyces bovis "The Ray fungus."

    Streptothrix hominis.

    Streptothrix buccalis.

Spirochætæ. Synonym: Treponemata.

    Spirochæta pallida or Treponema pallidum.

    Spirochæta vincenti or Treponema vincenti.

    Spirochæta of the mouth, as Treponema macro- and micro-dentium and others.

Higher Fungi:

    Ascomycetes.

        Blastomyces.

Oidium albicans.
Zygomycetes.
Rhizopus nigrans.
Sporothricum hominis.
Protozoa:
Sarcodina.
Endamœba gingivalis.
Flagellata.
Trichomonas. (Significance questionable.)
Spirochæta or Treponemata. (See bacteria.)

THE PYOGENIC COCCI

Staphylococci pyogenetes, or Pyogenic micrococci.
A. Micrococcus aureus or Staphylococcus pyogenes aureus.
B. Micrococcus citreus or Staphylococcus pyogenes citreus.
C. Micrococcus albus or Staphylococcus pyogenes albus.

The presence of micrococci in pus was in all probability first demonstrated by Pasteur[1] in 1880. The first to isolate and establish the relation of the most important member of the group to suppurative processes was Rosenbach[2] in 1884, who called the organism *Staphylococcus pyogenes aureus*.

This organism is widely distributed, being commonly found upon the external surface and in the oral and nasal cavities of man and many lower animals; it is not uncommonly found in the alimentary tract. When the tissue resistance is lowered, either locally, due to the action of some irritant; or generally, due to over-strain, malnutrition or some constitutional disease, this organism frequently invades the tissues at a point of least resistance, and produces either a circumscribed or diffused local infection. If the organism gains access to the circulatory system, from a localised infection, it is carried to various parts of the body giving rise to that type of general infection known as pyæmia. In some cases when the virulence of the organism is high, or the resistance of the infected individual is low, death may ensue within a short time and at autopsy no suppurative lesions (abscesses) will be found, the picture being that of a typical septicæmia. The *Micrococcus aureus* is frequently associated, as an important secondary invader, with lesions primarily

[1] Annales de Chimie et Physiologie.
[2] Archiv für Klin. Chirur., 1888.

due to some other organism, as in processes originally caused by the *Bacterium tuberculosum* or the *Streptothrix bovis.*

In the oral cavity this organism is encountered as the *primary factor* in superficial suppurative processes: Phlegmonous inflammation, localised abscesses, infections of the salivary glands, and in some cases of necrosis; also as a secondary invader in "pyorrhœa alveolaris;" in areas surrounding infected teeth; occasionally in apical and alveolar abscesses; in many cases of necrosis of the mandible and maxillary bone; in tubercular and actinomycotic lesions.

It is the organism almost constantly found in the peritonsillar abscess, that condition known ordinarily as "quinsy." Infections of the nasal cavity, antrum of Highmore and other accessory nasal sinuses and middle-ear infections, including mastoid disease, are not infrequently due primarily to the action of this organism. While the *Micrococcus aureus* and *albus* are the most common of the pyogenic organisms, the oral infections due to their action constitute but a very small percentage of all infections of the oral cavity.

The toxicity of *Micrococcus aureus* is due to an endotoxin; which endotoxin or another toxic substance produced by the organism (bacterial protein of Buchner) exerts a positive chemotactic action, *i.e.*, has the property of attracting leucocytes and producing pus. The phlogosine of Leber is probably identical with this substance; it also produces a leucocytotoxin known as a leucocydin. Some authors claim that the organism produces a soluble toxin, but from the fact that this so-called soluble toxin is only found in old cultures, one is led to consider it identical with the endotoxin, being liberated by the disintegration (autolysis) of the bacterial cells. The so-called hæmolytic properties of this organism are not due, as some claim, to a hæmolysin, but in all probability to the proteolytic enzyme produced by the members of this group.

It is of importance from a dental point of view, to note that the members of this group produce enzymes which have the property of acting on lactose, maltose, dextrose and certain other carbohydrates producing lactic, acetic and other mono-carboxylic fatty acids.

Protective and Curative Inoculations

Many attempts have been made to produce an anti-staphylococcus serum. Several such sera, obtained by inoculating animals with living and dead organisms and extracts of the bacterial cells, have been highly lauded by the investigators whose experimentation

led to their production. But it is a pertinent fact that none of them have been employed to any extent as therapeutic measures. When one states that immunity against these organisms is essentially opsonic and agglutinative, the reason for the therapeutic failure is at once manifest.

Bacterial vaccines, notably those prepared from that strain of the organism causing the infection, are of great value in treating and preventing the recurrence of infections due to the organisms of this group.

The infections in which the *Micrococcus albus* is found, either as a primary cause or a secondary invader, are rarely as severe as those in which the *Micrococcus aureus* is the causative agent. The action of this organism is of the same nature, and due to the same type of toxic agents, as that of the *aureus;* rarely is its virulence nearly so great as that of the latter. The *albus* is constantly found in *acne vulgaris;* frequently as a secondary invader of various other conditions; often in association with the *aureus.*

One strain of this organism, the *Staphylococcus epidermidis albus* of Welch, is found in the superficial layers of the skin, notably in the follicles, and has been demonstrated to be the causative factor in a large percentage of stitch abscesses.

Micrococcus albus vaccines are of value in treating localized infections caused by this organism.

The third member of this group, the *Micrococcus citreus*, so-called from the fact that in its growth on artificial media a lemon-yellow pigment is produced, is not so commonly encountered as the *aureus* and *albus;* some authors claim it to be non-virulent, while others make no mention of this organism when dealing with the group.

The degree of virulence of the *citreus* is as a rule about the same as the *albus.* Occasionally strains will be found possessed of a very high pathogenicity: such strains have been isolated from cases of pyæmia and carbunculosis. *Citreus* vaccines are employed in treating local infections caused by this organism.

Micrococcus Catarrhalis.—This organism is not infrequently found in the oral and nasal cavities of normal individuals. It is encountered in a small percentage of all cases of catarrhal inflammation of the oral and nasal mucous membranes; in some instances it has been found in groups of individuals presenting catarrhal infections; it has also been isolated from cases of bronchitis, pneumonia, and other infections of the mucous membranes. The pathogenic properties of this organism are not marked, and its ac-

tion in other than mild catarrhal conditions is usually that of a secondary invader, probably having little to do with the pathological process. The gingival trough is a normal habitat for this organism. (See Vol. 1, p. 221.)

In discharges from the oral and nasal mucous membranes the organism appears as a diplococcus, or singly and in groups, in the strands of mucus and fibrin, overlying the epithelial cells; and not infrequently within the protoplasm of the leucocytes. This fact has led to some confusion in differentiating this organism from the *Meningococcus* and *Gonococcus*, which is inexcusable in spite of the fact that they are all Gram negative, for the cultural and biochemical characteristics will readily enable one to differentiate between the *Meningococcus* and the *Catarrhalis*, and the fact that the *Gonococcus* will only grow on special media, renders the elimination of the latter organism comparatively easy.

The action of the *Micrococcus catarrhalis* is apparently due to the production of an endotoxin. Vaccines have been employed in treating conditions apparently due to this organism, but the unusually mild and acute character of the infections make it impossible for one to determine whether or not they are of any value.

Meningococcus.—This organism, discovered by Weichselbaum[1] and called the *Diplococcus intracellularis meningitidis*, is the cause of the infection known as epidemic cerebro-spinal meningitis. The latter term is in a sense an unfortunate one, as other organisms are capable of producing meningitis of an epidemic character.

The organism is spherical, occurring in pairs, usually within the polymorphonuclear leucocytes (pus cells), and is Gram negative, which makes it imperative to exclude *Gonococcus* and *Catarrhalis* infections when making a diagnosis. As stated when considering the *Micrococcus catarrhalis* this differentiation should not be difficult.

The organisms gain access to the tissues by way of the oral and nasal cavities, and the meningeal symptoms are preceded by nasopharyngeal symptoms, which may be manifest as a slight catarrhal disturbance or as a marked coryza and sore throat. An important fact to be noted is, that the organisms may lodge in sulci and crypts in the nasal and oral cavities, notably in the posterior nares, of healthy individuals; the latter constituting the so-called carriers, are manifestly an important factor in disseminating the disease.

Gonococcus.—This organism, the *Diplococcus of Neisser*, is the cause of gonorrhœal infection, usually affecting the urethral mucous

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[1] Grundriss der Pathologischen Histologie.

membrane in the male and the vagina and urethra in the female. The disease is readily transmissible to very few species of the lower animals. Infection of the eye, notably in infants (*Ophthalmia neonatorum*) is not uncommon. Nasal and oral infections are, on the contrary not frequently encountered; but when present it is manifestly of great importance that they be recognised.

The organism exerts a marked pyogenic action which becomes manifest as a severe inflammatory reaction of the mucous membranes, accompanied by a profuse discharge of a purulent exudate. In this exudate the micrococci will be found as diplococci, usually flattened on one side, within the pus cells. They are Gram negative, which fact aids materially in differentiating them from the ordinary pyogenic cocci. They can be isolated only by means of special media, these rendering their differentiation from the *Meningococcus* and *Catarrhalis* a matter of not more than twenty-four hours.

The Streptococci

From a dental point of view no single species or group of organisms, is of greater importance than the streptococci.

The older investigators divided the streptococci into the *Streptococcus longior et brevior* or *Streptococcus longus* and *brevis*, according to their occurrence in long or short chains. The fact was recognised that the organisms found grouped in long chains were possessed of the higher degree of virulence. Among these were classed the *Streptococcus pyogenes*, *Streptococcus erysipelatus*, which organism we now recognise as not being any different from the *pyogenes*, and *Streptococcus conglomeratus*, which is simply a strain of the *pyogenes* growing in conglomerate masses of closely intertwined chains. At this time that strain known as *Streptococcus anginosus* was not differentiated from the *pyogenes*. It is important to note that the above types have the property of hæmolysing erythrocytes, which fact has led to their being called the Hæmolytic streptococci.

The short chain streptococci include the organisms known as *Streptococcus viridans*, *Streptococcus mitis*, *Steptococcus fæcalis*, *Streptococcus saliavarius* and others.

Some strains of this latter group are of great importance to the dental practitioner; the most important, the *Streptococcus viridans*, being found in at least 90 per cent. of the oral cavities of human beings.

Some group with the streptococci the *Pneumococcus* and that

strain of the organism known as *Streptococcus mucosus capsulatus*. From certain viewpoints this is of advantage, notably when making isolations and group differentiations.

Owing to the dental importance of some members of the group we consider the subject of sufficient importance to admit of our discussing the group differentiation before taking up the properties of the various organisms.

Streptococci grouping and methods of differentiation have been subjects of discussion and investigation for twenty years, the details of which will be found in works on the subject of pathological bacteriology.

The methods of differentiation are based principally upon the morphological and biochemical features. In connection with the former, such characteristics as the type of chain formation and whether or not capsules are produced; as regards the latter, the action on various carbohydrates, on hæmoglobin, and the effect of bile on the life and growth of the organism. Within the last few years differentiation by the complement-fixation and agglutination reactions have aided in separating these organisms.

In discussing this phase of the subject, we will assume that several organisms have been isolated and studied as regards their ordinary morphological characteristics, cultural and biochemical features. On making such studies, one is forcibly impressed by the marked similarity of the various cultures. One group will not ferment lactose: the type of this group is *Streptococcus equinus*.

The next proceeding will be to culture the organisms fermenting lactose in a medium (sugar-free broth or serum water) containing 1 per cent. of inulin. If the medium is fermented, the organism is a member of the *Pneumococcus* group. This can be confirmed by culturing in bile medium. The various strains of the pneumococcus being bile-soluble, are destroyed, while the streptococci will develop luxuriantly.

The organisms of the group incapable of fermenting inulin and not bile soluble, namely the *Streptococci*, are cultured in blood-agar plates, when the type of reaction admits of their being separated into three groups: (1) Those hæmolysing erythrocytes, which phenomenon is manifest by the appearance of a clear zone surrounding the colonies; these are the *Hæmolytic streptococci;* (2) Those producing methæmoglobin, which reaction is manifest by the appearance of a greenish colouration of the colony and a greenish zone in the medium immediately surrounding it; this group includes the *Strep-*

tococcus viridans and the *Streptococcus salivarius;* (3) Those exerting no action on hæmoglobin or erythrocytes, as the *Streptococcus fæcalis* and *Streptococcus lactis.*

The organisms of the hæmolytic type may be differentiated by culturing in a broth or serum-water medium containing the glucoside salicin; the ones fermenting salicin, with acid production are strains of the *Streptococcus pyogenes*, those not fermenting salicin the *Streptococcus anginosus.*

Of the group producing methæmoglobin, the *Streptococcus viridans* ferments salicin while the *Streptococcus salivarius* does not. The *Streptococcus fæcalis*, of the indifferent group, ferments the glucoside and the *Streptococcus lactis* reacts negatively.

Streptococcus Pyogenes.—This organism is spherical in form and found grouped in long chains. Such chains are always manifest when preparations made direct from pathological lesions are examined, and usually in cultures, especially when cultured in liquid medium or in the water of condensation of blood-serum or agar slants (Fig. 368).

This organism is found as the primary invader in a variety of local infections, some circumscribed, others diffuse as in erysipelas, and not infrequently as a secondary invader in processes primarily due to some other organism.

It is the type usually found in the septicæmia following childbirth (puerperal septicæmia), in which case the organism gaining access to the uterus, by way of the vagina, is absorbed and carried to various parts of the body. When this organism gains access to the general circulation from a localised infection (focal infection), the resulting septicæmia is generally of a fulminating character, causing death within a comparatively short time; some few cases recover. Cultivation on artificial media quickly reduces the virulence of this organism. On the other hand, the virulence may be greatly enhanced by passage through animals, notably rabbits, and undoubtedly the same increase in virulence is true of organisms developing in acute infectious processes.

This organism is the one usually concerned in follicular tonsillitis, in some acute gingival and buccal infections, occasionally in infections of the salivary glands and diffuse cellulitis of the oral tissues. In a certain percentage of the cases of that symptom complex known as Ludwig's angina the *Streptococcus pyogenes* is found; it is encountered in some cases of necrosis of the mandible and maxilla; and also in a certain percentage of antral and frontal sinus infections. Contrary to the statements occasionally made to the effect that this

organism is common in apical abscesses, it is but rarely found in either apical or alveolar abscesses. Neither is it commonly found in "pyorrhœa alveolaris."

This organism may be the sole cause of pneumonia and not infrequently secondarily invades the lung in cases of pneumococcus pneumonia. It also frequently plays an important part by secondarily invading a pulmonary tubercular abscess, in that it adds to the toxæmic condition and induces the breaking down of tissue leading to cavity formation.

The toxic products of the organism are, an endotoxin, a soluble toxin, and a hæmolysin.
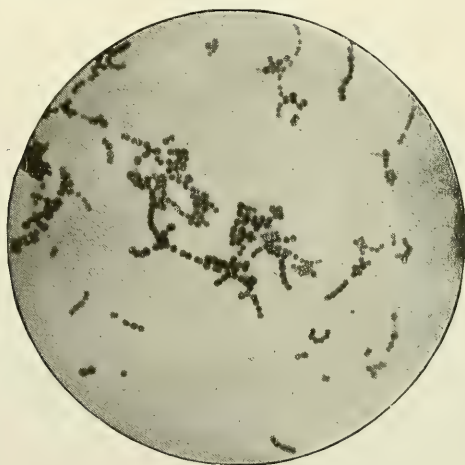
FIG. 368.—*Streptococcus longus* (*pyogenes*), from a fatal case of pyæmia. Magnified 1,000 times. (*Trans. Odonto. Soc. of Great Britain.*)

Anti-streptococcus sera of various types have been employed in treating different types of streptococcus infection. On the whole the results have been extremely disappointing. Autogenous streptococcus vaccines are of value in treating certain acute and subacute infections. They may be employed in general infections, but the results obtained are not of such a nature as to cause one to consider them among the important therapeutic measures. The so-called stock polyvalent streptococcus vaccines are, in the writer's opinion, of absolutely no value.

Streptococcus anginosus.—This organism is closely related to the *Streptococcus pyogenes*, occurring in the same long chains, exerting a hæmolytic action, but differing from it in the fact that salicin is

not fermented. As a rule it causes coagulation in milk. The latter feature however is not constant.

This strain is found in a large percentage of cases of so-called epidemic septic sore throat, and is also encountered as a secondary invader in many cases of scarlet fever. Extensive epidemics of septic sore throat have been reported from time to time in various countries, and in America numerous epidemics have occurred. This type of sore throat, affecting the fauces and tonsils, is very severe and accompanied by marked constitutional symptoms, at times due to toxæmia. In other cases, in addition to this, the organism invades the tissues *viâ* the circulation, producing a septicæmia
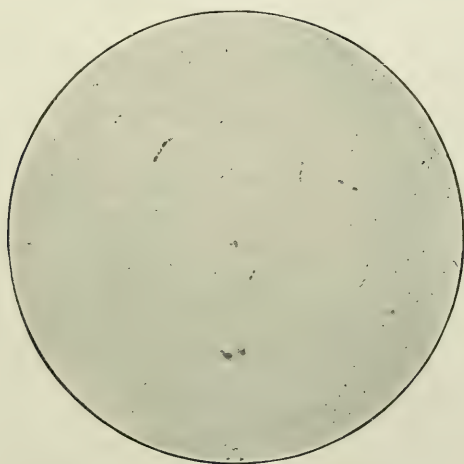
FIG. 369.—*Streptococcus brevis,* from a healthy mouth. Magnified 1,000 times.
(*Trans. Odonto. Soc. of Great Britain.*)

which has resulted in a comparatively high percentage of deaths. The organism giving rise to the epidemic may be disseminated by means of milk from cows affected with mastitis; or by individuals who are infected with the organism handling the milk, or receptacles in which it is collected and distributed; in other epidemics the organisms are evidently disseminated in some other manner.

Vaccines and sera are of little or no value in treating this infection.

Streptococcus Viridans.—This name is applied to that short chain *streptococcus* producing methæmoglobin when cultured in blood media, and fermenting salicin with acid formation.

This organism is constantly encountered in the oral cavity, not

only in pathological conditions but also in healthy individuals, even of those who exercise great care in cleansing their mouths (Fig. 369).

It is encountered as the primary cause or as a secondary invader in the majority of oral infections, in fact in practically all superficial infections. In some cases its presence is of no moment, in others it has a marked detrimental action on the tissues. The presence of this organism in apical abscesses is almost constant, and it is found in a great percentage of alveolar abscesses. In many cases of antral and frontal sinus infections the *viridans* is encountered either alone or in association with some other organism. Manifestly it is present in many cases of necrosis.

When this organism gains access to the circulation from a focal infection it is carried to all parts of the body. It rarely possesses a marked degree of virulence and therefore does not often give rise to a fulminating, but rather to a low-grade septicæmia. In many cases when it is disseminated throughout the body, the pathological action is not manifest as a general infection but localised in one part, and such conditions as arthritis, synovitis, neuritis, endocarditis, etc., arise. Very often clearing up of the focal infections leads to the disappearance of all symptoms. The reason for this is manifest; the organism possesses a low degree of virulence and the bodily defenses are enabled to prevent a general action on the tissues. Many of the organisms are destroyed; those remaining in a locality of lowered resistance are enabled to develop and exert a detrimental action on the tissues. In many cases when these organisms produce a low-grade septicæmia, the condition may be cured or may lead to the death of the patient after a somewhat prolonged illness.

Streptococcus Salivarius.—This strain of the short chain streptococci may be encountered in the same type of infection as the *viridans;* it is however, as a rule possessed of but a very low degree of pathogenicity.

Pneumococcus or Diplococcus Pneumoniæ.—This organism in cultures cannot always be differentiated readily from the short chain *streptococci*, as it frequently occurs in short chains and reacts culturally in about the same manner. The various types of this organism ferment lactose and inulin: all are bile soluble. Within the last few years various types have been separated; types differing one from the other sufficiently to admit of their being differentiated biologically; pathologically their action is much the same. For information on this and the general phases of pneumococcus infec-

tion, the student is referred to recent works on pathological bacteriology.

When stained by appropriate methods in sputum, catarrhal exudates, pus, blood and tissue fluids, the organism appears as a diplococcus surrounded by a definite capsule. At times three or four elements will be enclosed in one capsule; one type called the *Streptococcus mucosus capsulatus* occurs in long chains surrounded by a distinct capsule.

The *Pneumococcus* is not infrequently found in the oral cavities of healthy individuals; therefore one might expect to find it in various pathological conditions. In some cases of ulcerative stomatitis, in which there are present small, often painful, superficial ulcers, the *pneumococcus* is present as the primary infective agent; it is found in many cases of "pyorrhœa" and other gingival infections. If alveolar abscesses be detected soon after their formation and proper precautions be taken in opening and examining their contents, the *pneumococcus* will be frequently found in pure culture; it is however, rarely found in apical abscesses. Occasionally the *pneumococcus* in association with *streptococci* and other organisms, is encountered in necrosis. Not infrequently it is found in antral infections. Should the organism gain entrance to the general circulation, a pneumococcus septicæmia may result. There are on record cases of pneumococcus meningitis; in some of these cases the organisms in all probability gain access to the cranial cavity by way of the nose or naso-pharynx.

This organism has been demonstrated in not a few cases of acute catarrhal infection of the upper air passages as apparently the only organism present capable of causing such infection. Pneumococcus conjunctivitis is a well-recognised infection.

Bacillus diphtheriæ, or more properly speaking, *Bacterium or Mycobacterium diphtheriticum,* the cause of the disease known as diphtheria, is occasionally encountered in infections of the oral mucous membrane. The fact that such infections are not more common is somewhat surprising when one considers the frequency of diphtheritic infection of the tonsils and pharynx.

This organism occurs in the form of short or long rods presenting clavate or cuneate swellings. In stained preparations the protoplasm, instead of appearing homogeneous or finely granular, is barred or striated, in many cases containing metachromatic granules which, when stained by appropriate methods, assume a colour different from the other constituents of the cell, indicating that chemically they are of a different nature from the rest of the protoplasm.

When a given strain of this organism is cultivated on various kinds of media and examined, one is forcibly impressed with the marked variations in morphology exhibited; variations in the reaction of the medium: presence of substances exerting a detrimental action on the organism. Hypo- or hypertonicity seem to be the determining factors in these morphological variations. The bizarre forms are known as involution forms; under certain conditions of cultivation the organisms present buds and not infrequently short and long branches which branches are frequently swollen at the ends.

The pathogenic action of the organism is due solely to a soluble toxin; as a means of combating diphtheritic infection an antitoxic serum is employed.

From a dental viewpoint it is of importance to note that healthy individuals may be carriers, the organism developing in the crypts of the tonsils and sulci in the naso-pharynx.

There are several types of organisms morphologically like the *Mycobacterium diphtheriticum*, which are frequently encountered in the oral cavity, some colourless, others producing coloured growths on artificial media. These are grouped under the general term of *pseudo-diphtheritic organisms*. Pathologically from our viewpoint they are of very little import. Some strains may be concerned in mild catarrhal conditions of the oral and nasal mucous membranes. The chromogenic types are commonly found in milk.

The import of this group rests on the fact that they resemble the diphtheria organism morphologically, and, to a great extent, culturally, and must be differentiated from the latter in suspicious cases. In no case do they induce infection in any manner resembling diphtheria.

Bacillus tuberculosis, or *Mycobacterium tuberculosis*, is the cause of the various tubercular infections as: pulmonary tuberculosis, general miliary tuberculosis, glandular tuberculosis, tuberculosis of the skin, etc. This organism is one of the few that can be readily and positively diagnosed in stained preparations of sputum, pus, etc., the diagnosis depending on the fact that one of the constituents of the cell of the organism is a peculiar wax which gives to it "acid-fast" or acid-resisting properties. It stains with difficulty, but when once stained, retains the stain tenaciously, resisting to a marked degree the action of decolourising agents. Therefore when material is stained with an intense red dye such as carbo-fuchsin; treated with decolourising agents such as 30 per cent. nitric acid, 5 per cent. sulphuric acid or acid alcohol; washed and counter-stained with methylene blue, the

presence of the *B. tuberculosis* is indicated by the finding of red rods, straight or curved, usually beaded, in a field, all other elements of which are stained blue.

Tubercular infections are of great importance to the dental practitioner. In the first place in pulmonary tuberculosis the organisms are present in the sputum, and manifestly in the oral cavity. Tubercular infections of the tonsils and oral mucous membrane are not common but are occasionally encountered. (See page 355). Tubercular infection of the salivary glands are occasionally seen, as is tubercular caries of the mandible and tubercular antral infections. One type of tubercular infection, the lesions of which are not infrequently mistaken by the dentist as being due to syphilitic infection, is that condition of the nose and face commonly known as *Lupus vulgaris*. Not infrequently this results in a destruction of tissue to such an extent as to cause marked disfigurement, not only by destroying the soft tissue of the nose but bony tissue as well. In all of these conditions the organism may be readily demonstrated in the tissue by appropriate methods of staining.

Bacillus Mallei, Glanders Bacillus, or *Mycobacterium Mallei*, is the causative agent in the disease known as glanders. This disease is primarily a disease of horses, mules and asses, but is occasionally transmitted to other domestic animals and man. There has been much discussion as to the portal of entrance of the organism, but undoubtedly in a certain percentage of cases, it gains access by way of the nasal and oral orifices.

Veterinarians, hostlers and others coming in contact with horses are most often infected, and present oral and nasal inflammatory lesions due to the action of this organism.

Bacillus anthracis or *Bacterium anthracis* is a rod-shaped organism, non-motile, occurring in chains and forming endospores. It is the causative agent in the disease known as *Anthrax, Charbon*, or *Malignant pustule*. This infection, primarily a disease of cattle and sheep, may be transmitted to man. The organism enters the tissue through an abrasion, or by way of the nasal and oral cavities, being taken into the lungs with dust, and into the intestinal tract with food and drink. It produces a marked œdematous inflammation at the point of entrance, and fulminating septicæmia when, after gaining access to the circulation, the organisms are carried to all parts of the body. The pulmonary type of anthrax infection is frequently called "wool-sorter's disease."

Bacillus mucosus capsulatus, Friedländer's bacillus, or the *Bac-*

terium mucosum capsulatum is a capsulated, non-motile, rod-shaped organism which does not form endospores.

Classed under this group are several types varying not at all morphologically and but very little culturally. They are differentiated one from the other essentially by their action on certain carbohydrates. One type, the *B. rhinoscleromatus*, is found in the disease known as rhinoscleroma; a second, the *B. œzenæ*, occurs quite constantly in cases of fœtid catarrhal inflammation of the nose. The *Bacterium mucosum capsulatum* is very frequently encountered in the oral cavities of healthy individuals; it exists also in a variety of pathological conditions, probably rarely as a primary cause, but rather as secondary invader. Some claim that this organism is the cause of *scleroma linguæ*.

Bacillus aërogenes capsulatus, the so-called gas bacillus of Welch is found in the intestinal tract of man and lower animals. This organism is the cause of gas gangrene, the gas production being due to its ability to split up complex substances, notably carbohydrates, with the formation of simpler compounds, among which are certain gases.

This same organism has been described by various authors who apparently knew nothing of the work of Welch. The *Bacillus perfringens*, the *Bacillus of Ghon* and *Sachs*, *Bacillus phlegmonous emphysematosæ* of Fraenkel and others are identical with the bacillus of Welch.

This organism is a strict anaerobe, occurs singly and in chains. When found in the tissues and stained appropriately, it always presents a distinct capsule. It is non-motile, forms endospores and reacts positively to Gram's stain. Its natural habitat is evidently the soil. This organism has been encountered not only in the oral cavities of normal individuals, but in pathological conditions where individuals have extensive injuries to the face and oral tissues, notably when soil and excrementitious substances contaminate the wounds. The danger of such infection should be realized and proper precautions taken against such untoward complications occurring.

It has been claimed that Miller first discovered this organism as existing in the oral cavity.

Bacillus Tetani.—This organism, a strictly anaerobic, spore-forming motile rod, is the cause of the disease known as tetanus, frequently called "lockjaw."

While tetanic spasm of the jaws is a prominent symptom of this

disease it is rare to find the focus of invasion in the oral cavity, although the organism is undoubtedly frequently present here as well as in the intestinal tract of healthy individuals.

This organism is constantly encountered in the soil in some localities, and gains access to the tissues through wounds, developing at the point of invasion, producing a soluble toxin which is carried throughout the tissues by the circulation, the organism remaining at the local lesion.

There are other anaerobic organisms found in the soil which may be found in the oral cavity. Some are pathogenic but, rarely, if
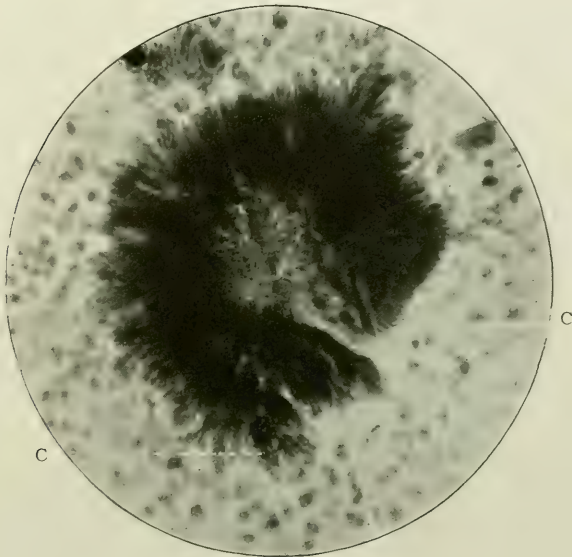
FIG. 370.—*Streptothrix actinomyces.* From section of a tongue of an ox. Stained by Gram's method, counterstained with eosine. Magnified 800 times. c. Club-shaped forms.

ever, concerned in oral infections. Some are organisms of the malignant œdema type, others, while not of a pathogenic nature, may play an important rôle in dental caries.

Streptothrix bovis, variously known as *Streptothrix actinomyces,* *Actinomyces bovis, Discomyces bovis,* the *Ray fungus,* occurs as long thread-like, interlacing filaments, presenting a true branching and at times, small spherical bodies, the true significance of which is still a mooted question, some writers claiming they are spores or conidia and others denying this.
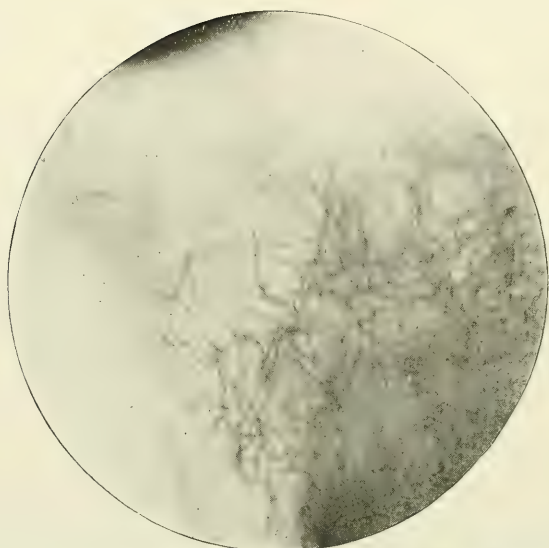
FIG. 371.—The same. From a culture. Magnified 800 times.
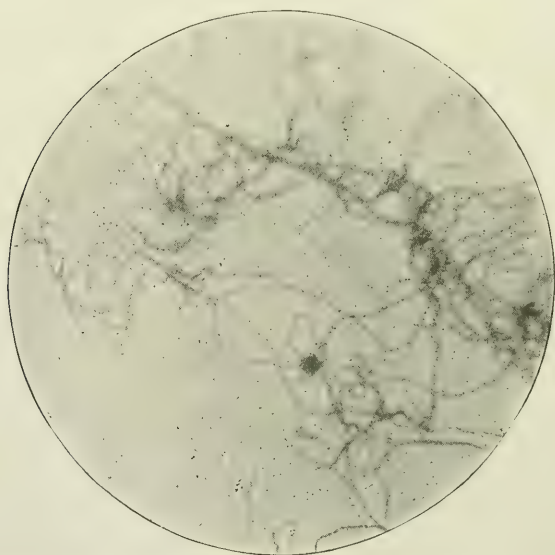
FIG. 372.—*Cladothrix dichotoma*. Stained by Gram's method. Magnified 800 times.

When examining pus, sputum, and the tissues of infected animals, small yellowish or whitish opaque granules will be found, usually about the size of a millet seed. If these granules are crushed, stained and examined microscopically, the organism will be readily demonstrated as long entangled branched filaments, usually terminating in bulbous ends (clubbed). These filaments in general arrangement radiating from the centre give rise to a radiate mass which led to the organism being called the "ray fungus." In tissue sections the radiate colonies are readily demonstrated.

This organism is the cause of "lump-jaw" and "wooden tongue" or actinomycosis of cattle, but while it is frequently localised in the

FIG. 373.—*Cladothrix* from mouth direct. Stained by Gram's method. Magnified 1,000 times. (*Trans. Odonto. Soc. of Great Britain.*)

mandible, the organism in many cases invades the other tissues. In man, oral actinomycosis is occasionally encountered, not only as lesions of the mandible and maxilla, but the soft tissues as well, notably the tongue.

The name *Streptothrix hominis* has been given to some organisms of this group from human sources. Whether or not this is a type different from the *bovis* is an open question. One type found not infrequently in the oral cavity has been named the *Streptothrix buccalis*. This organism is encountered in apparently healthy individuals, usually those whose mouths are not kept clean. It has been encountered in the tonsillar crypts, in the so-called mycosis of the tonsils; and in deep tonsillar lesions. Similar organisms have

been found in a few of those cases of chronic bronchitis in which there is a very profuse expectoration of a muco-purulent material (bronchorrhœa).

Members of the streptothrix group have frequently been mistaken for the *Cladothrix dichotoma*, an organism belonging to the genus *Cladothrix* or *Sphærotilus* of the family of *Chlamydobacteriaceæ*. Their cultural characteristics are similar; but by appropriate methods of staining, the latter organism will be found to present a distinct sheath and the apparent branching to be not a true, but a false branching. This organism is occasionally found in the oral cavity but is of no pathological significance (Fig. 373).
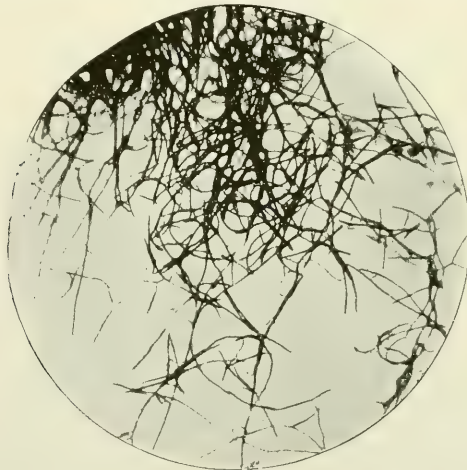
Fig. 374.—*Cladothrix*. Forty-eight hours' cultivation in agar. Magnified 500 times. (*Trans. Odonto. Soc. of Great Britain.*)

Bacillus fusiformis or *Mycobacterium fusiformi* was first discovered by Vincent in cases of hospital gangrene. This organism is almost invariably associated in pathological conditions with the *Spirochæta vincenti* (Treponema vincenti). Both are anaerobic organisms and are difficult to isolate, but may be readily demonstrated in the exudate from pathological conditions in which they are present, making a diagnosis not difficult. Some claim that they are but pleomorphic forms of one organism. This view, however, cannot be accepted. The *Bacillus fusiformis* is a non-motile, rod-shaped organism, slightly enlarged in the middle and pointed at the ends. The *Treponema vincenti* is a long slender, curved, wavy or sinuous organism somewhat thickened at the middle and gradually taper-

ing out to a thin flagella-like filament at the ends. When examined under dark field illumination the organism will be found to present active sinuous and wavy movements.

These organisms are constantly found in that tonsillar infection known as Vincent's angina; in certain types of localised and spreading ulcerative stomatitis (especially along the gingival border); in many cases of "pyorrhœa alveolaris;" in some cases of that diffuse cellulitis of the floor of the mouth, frequently called Ludwig's angina (Angina Ludovici); occasionally in infections of the salivary glands, notably the submaxillary and sublingual; constantly in gangrenous stomatitis or noma (*Cancrum oris*). The infection called "trench mouth" is an ulcerative stomatitis, the causative agents of which are the *Bacillus fusiformis* and the *Treponema vincenti*. Infections due to these organisms occur in various other parts of the body.

Spirochæta pallida or *Treponema pallidum*, the specific infective agent of syphilis, will be found in the primary and secondary oral lesions of syphilis, when material from the lesions, carefully obtained, is examined by dark field illumination or in preparations stained by special methods.

Under dark field illumination it is seen as a spirally curved organism turning rapidly upon itself and presenting a rotary and sinuous movement.

When stained by appropriate methods the organism appears as spiral cells from 8 to 14 μ in length, tapering to a slender flagella-like extremity at each end. The turns of the spiral are close and regular. It cannot be demonstrated by the ordinary methods of staining; other spirochætes have been mistaken for the *pallida*, but the danger of one who has had some experience of making such an error is slight, the other types varying in size, form and arrangement of the spirals to such an extent as to render their elimination an easy matter. Several spirochætes have been described as frequently occurring in the mouth, one author, basing his contention on morphological grounds alone, separated them into eight species. This is undoubtedly erroneous. The *Treponema microdentium*, formerly called *Spirochæte dentium*, and the *Treponema macrodentium*, formerly called the *Spirochæte buccalis* and the *Treponema vincenti* are the ones most commonly found. They are readily demonstrated in stained preparations.

Oidium Albicans—Saccharomyces Albicans or *Endomyces Albicans.*—This organism belongs to the same group of cryptogams as

the yeasts, namely the Eumycetes or true fungi, order of Asco-
mycetes.   The oidium evidently belongs to the family *Endomyces*.

This organism is the cause of mycotic stomatitis, the condition
variously known as "thrush."   Some have called the condition
oidiomycosis, rather unfortunately, as this name has also been
applied to blastomycotic dermatitis.

If a small particle of one of the characteristic milk white, friable
patches from the oral mucous membrane be spread upon a slide,
stained and examined, long filaments, septated into oval or rod-
shaped elements, will be found.   There are frequently present numer-
ous spherical or oval bodies, often containing a smaller spherical
body, known as the ascospore.
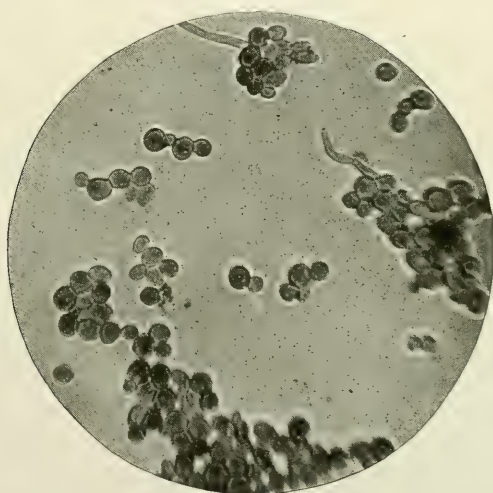
FIG. 375.—*Oidium albicans*.   From a culture.   Magnified 600 times.

Several kinds of *Yeasts* have been encountered in oral infections.
Whether or not they are distinct types is a question, as the descrip-
tions given are not sufficiently complete to admit of absolute differ-
entiation.   From the similarity of the lesions produced, the majority
are undoubtedly identical with the *Blastomyces dermatidis* of Gil-
christ, first found in a peculiar type of chronic dermatitis.   This
organism is occasionally encountered in a chronic ulcero-granulo-
matous condition of the oral tissues, which has in some cases
been mistaken for carcinoma until a microscopic examination cleared
up the diagnosis.   In the tissues the organism appears as oval or
spherical cells, averaging from 10 to 12 μ in diameter; the

majority of the cells being about 5 μ in diameter, while some elements are much larger.

The cell, by appropriate methods of staining, presents a thick membrane surrounding the central protoplasm. These cells not infrequently, owing to the fact that they multiply by budding at least in one stage of their development, occur in pairs of unequal size.

A Yeast, the *Saccharomyces nigrans*, has been found in some cases of *Linguæ nigra* or black tongue. When examined microscopically the cells appear as small oval bodies, occurring singly and in pairs. Occasionally three will be grouped together; one cell, the parent cell, will be larger, the attached cells smaller than those occurring singly. In cultures this organism produces a black pigment. The organism commonly found in the mycotic type of *Linguæ nigra* is however not a yeast but the *Rhozopus nigrans*, a black mould closely related to, some claim identical with, the *Mucor nigrans*. In stained preparations this organism may be demonstrated among the epithelial cells as long filaments septate and often branched. At times the characteristic oval or round spores will be found in such preparations.

Sporothrix Infections.—The organisms causing the so-called Sporothricosis apparently belong to the *Ascomycetaceæ;* the type organism is the *Sporothrix schencki*. This organism invades the tissues at some local point, usually where their resistance has been lowered, and gives rise to peculiar hard, indurated subcutaneous and submucous lesions in man. These lesions frequently break down and abscesses are formed which are usually chronic. The organism may invade the buccal, pharyngeal and laryngeal mucous membranes. In the purulent exudate the organism is found among the pus cells as yeast-like cells, oval and fusiform in character; at times a filament will be found with groups of the oval bodies (spores) arranged about the end. The organism is readily isolated and cultivated on artificial media. In such cultures the morphological characteristics of the organism vary quite markedly from the characteristics of the organism as found in the tissues.

THE FILTERABLE VIRUSES

There are certain diseases having all the characteristics of infections, in which all attempts made at isolating the causative agents or even demonstrating such agents microscopically, have met with failure. Further investigation demonstrated the fact that when the material, macerated and diluted, was passed through a so-called

germ-proof filter, the filtrate when injected into suitable animals gave rise to the same type of infection as that in which the original material was found. Several such infections exist, of which the one of most importance to the dentist is "foot-and-mouth disease," or aphthous fever. While primarily a disease of cattle, sheep, goats, and pigs, the infective agent, during epidemics, not infrequently gains access to the oral cavities of human beings directly from infected animals, infected milk, etc., and causes a severe aphthous inflammation.

Endamœba Gingivalis (Gros).—This protozoon was first described by Gros in 1849, and named *Amœba gingivalis;* since then it has been observed by others and variously named *Amœba buccalis*, *Amœba dentalis* and *Entamœba buccalis* (Fig. 376).

The organism was frequently observed in microscopic preparations from dirty mouths, pyorrhœal infections, etc. However no pathological significance was attached to its presence until Smith and Barrett,[1] after a study of a group of cases of "Pyorrhœa alveolaris," announced the fact that the organism was found in a large percentage of cases of infected gingival tissues. They ascribed to it an important etiological relationship as regards pyorrhœal infections.

The organism may be demonstrated in fresh preparations by mixing the material with a drop of salt solution on a warm slide, applying a cover glass and searching the slide for motile organisms with an 8-mm. lens. The preparation must be kept warm, therefore the search is best made with a microscope fitted with a warm stage.

The organism may also be readily demonstrated in dried preparations by staining with appropriate stains, as Giemsa's method or with carbol-fuchsin followed by methylene-blue.

In fresh preparations the *Endamœbæ*, averaging about 30 μ in diameter are quite actively motile, presenting an irregular outline, due to throwing out of pseudopodia. In the quiescent stage they are usually circular or oval, the ectoplasm is hyaline, while the endoplasm is granular and usually contains bacteria, disintegrated erythrocytes and leucocytes. The nucleus can rarely be seen in fresh preparations. Vacuoles are usually present, some empty while others contain inclusions of different sizes and shapes. When stained, the ectoplasm usually appears to be finely granular in structure; the endoplasm coarsely granular and frequently alveolated. Inclusions of various sizes and shapes are usually found within the endoplasm.

[1] "The Protozoa of the Mouth in Relation to *Pyorrhœa Alveolaris*," "Dental Cosmos," Aug., 1914.

The nucleus is circular or oval with a centrally located chromatin mass.

ADVENTITIOUS BACTERIA OF MOUTH

There are other organisms found in material from the mouth which have not been encountered in any other place, although their original habitat is undoubtedly some place in nature, as soil, water, etc. Certain of these organisms are bacteria, others higher fungi; they
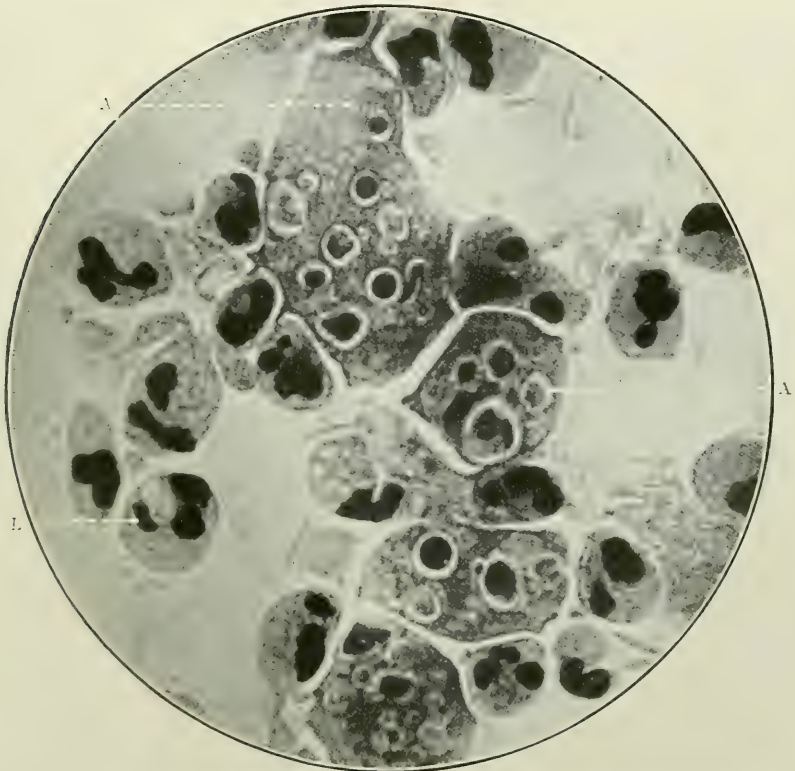
FIG. 376.—*Endamœba gingivalis.* A. Amœbæ; L. Polymorphonuclear leucocyte. (*Photomicrograph by courtesy of Dr. Percy R. Howe.*)

have been named and described by various early investigators, notably Miller,[1] and Vicentini,[2] later by Leon Williams[3] and Goadby.[4]

[1] "Microörganisms of the Human Mouth," 1889.
[2] *International Journal of Microscopy and Natural Science*, 1894-95.
[3] "Contributions to the Bacteriology of the Human Mouth," Dental Cosmos, 1899.
[4] "Mycology of the Mouth," 1903.

Some of the organisms have been identified with others occurring more or less widely disseminated; others, owing to the fact that they were not described in sufficient detail cannot be recognised as distinct species today. Some of these organisms are constantly encountered in stained preparations from the mouth, notably in the case of individuals who do not exercise proper care in cleansing the oral cavity; also in the material which collects on the teeth during sleep, and in the so-called mucinous plaques (bacterial plaques) which form on the surface of the teeth. Such organisms are often found microscopically in many pathological conditions, not as causative factors but as accidental contaminations. Some can only be recognised by their morphological characteristics, owing to the fact that they have resisted all attempts directed toward their isolation and study in artificial media. Possibly some have been isolated and are known under other names, it being a well-known fact that certain organisms differ markedly in form under varying conditions of development, as it were in different phases of their life cycle.

For the purpose of illustration, suppose we refer again to the microscopic study of material of various kinds from the oral cavity. The most important of the organisms concerned in pathological conditions are: various types of cocci, rod-shaped organisms, spiral and thread forms; these have been isolated and studied in cultures. There are others which are readily isolated which have no pathological significance, although they may play a part in dental caries. Among such organisms are the following:

Sarcina alba, lutea and aurantica.—These develop readily on artificial media, producing respectively no pigment, a yellow, and an orange pigment. They divide in three planes, the cells remaining approximated, resulting in the formation of cubical masses. These cocci are quite large, 1 to 1.5μ in diameter, non-motile, non-spore forming, and are positive to Gram's stain.

Iodococcus vaginatus of Miller which he claimed is found in all unclean mouths occurs singly, or in short chains, and gives the iodine reaction. The identity of the organism has never been established: its importance is probably only historical.

Micrococcus tetragenus or *Sarcina tetragena* is observed in oral material, appearing as small cocci from 0.6μ to 0.8μ in diameter occurring in groups of four. This organism is at times noticed in pathological conditions as a secondary invader.

Of the rod-shaped organisms encountered, those worthy of men-

tion, beside the pathogenic organisms already considered, are the following:

Bacillus buccalis maximus, which presents as jointed threads 10 to 50μ long and 0.5 to 1.5μ wide, frequently straight, at times curved, positive to Gram's method. Flagella are present. Goadby has isolated and described an organism under this name which apparently belongs to the *Mesentericus* or *Subtilis* groups of the ordinary spore-forming soil organisms. *Leptothrix buccalis* of Vignal[1] and *Leptothrix buccalis maxima* of Miller are probably identical with the above.
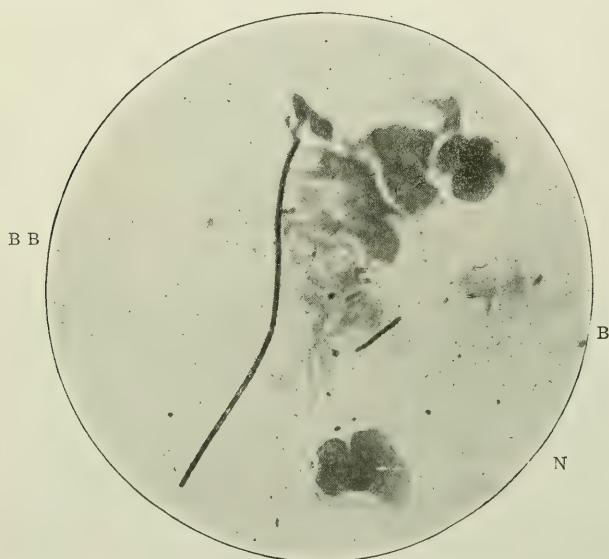
Fig. 377.—*Bacillus buccalis maximus.* Preparation similar to Fig. 462. Magnified 800 times. b.b. *Bacillus buccalis maximus;* b. Large rod-shaped form; n. Nucleus of partially-digested epithelial cell.

Various types of the so-called spore-forming organisms of the soil are met with in stained preparations. These can be readily separated in cultures. Among them we find the *Bacillus subtilis; Bacillus mesentericus fuscus*, which is identical with the *Bacillus gangrenæ pulpæ* of Arkovy;[2] *Bacillus mesentericus ruber* and others.

Non-sporulating rod-shaped organisms of various types may be noted, as the *Bacillus proteus vulgaris, Bacillus coli*—organisms

[1] Archives de physiol., normal path., 1886.
[2] *Vierteljahresshrift für Zahneilkunde*, Jahrgang, xiv.

FIG. 378.—Various forms of micro-organisms from the mouth direct. The fine threads are Miller's *Leptothrix innominata:* the thick chain is *Bacillus buccalis maximus.* (From Goadby's "The Mycology of the Mouth.")

FIG. 379.—*Leptothrix buccalis maxima,* and *Bacillus buccalis maximus.* From approximal surface of a tooth. Magnified 1,500 times. (*Photomicrograph by Leon Williams.*)

FIG. 380.—*Spirillum sputugenum* from mouth direct. Stained with gentian aniline violet. Magnified 1,000 times. (*Trans. Odonto. Soc. of Great Britain.*)
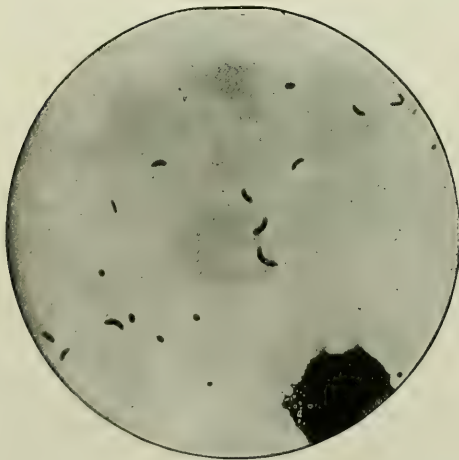
FIG. 381.—*Spirillum sputugenum* (comma forms) from mouth direct. Stained and magnified as in preceding Fig. and from same source.

that cannot possibly be recognised except by isolating and studying their characteristics.

Members of the Spirillum group are found in a large percentage of cases. The organism known as the *Spirillum* of Miller, *Spirillum sputugenum*, or *Microspira Milleri*, which occurs as short slender comma-shaped cells, is not infrequently encountered. The only way in which this organism can be differentiated from other *Microspiræ* is by isolation and culturing.

The important *Spirochætæ* or *Treponemata* have already been considered.
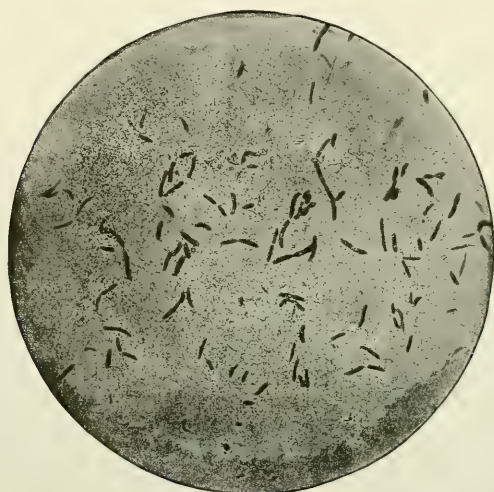
Fig. 382.—*Spirillum sputugenum*, freshly isolated from the mouth. Spiral forms not yet well developed. Magnified 1,000 times. (*Trans. Odonto. Soc. of Great Britain.*)

Strikingly interesting organisms, probably more interesting than important, are certain of the *Chlamydobacteriaceæ* or sheath organisms. The *Cladothrix dichotoma* is occasionally observed, but cannot be distinguished from certain other thread-like organisms, in preparations made direct from the oral cavity. In cultures it is readily differentiated from all, excepting the *Streptothrix buccalis*, and from this by careful morphological study. It occurs in the form of long threads, straight or curved, apparently branched. It is of no pathological significance. The *Leptothrix innominata* of Miller occurs in slender threads from 20 to 30μ in length usually straight, at times curved, rarely segmented, frequently presenting small gran-

ules. This organism has never been isolated and identified as such. It presents a sheath when stained by appropriate methods, therefore it would appear to be a true *Leptothrix*.

The so-called *Leptothrix racemosa* of Vicentini is very frequently discovered in the oral cavity, and has always attracted a great deal of attention on account of its striking form. Vicentini at one time looked upon this organism as the parent, as it were, of all bacteria found in the sputum. This of course was many years ago when the theory of pleomorphism was receiving a great deal of undeserved attention. The organism has never been isolated. Where it should actually be placed in the cryptogamic classification is not known; it is not a true *Leptothrix*. The writer has suggested on several occasions that it be classified for the present with the *Fungi imperfecti*.

It has been studied in detail morphologically by Vicentini and Leon Williams. Owing to the fact that the organism is of considerable historical and biological interest, if probably non-important pathogenically, the description given in the first edition of this book, reading as follows, is appended.

"Considerable interest has lately attached to the *Leptothrix racemosa*, an organism first described by Filandro Vicentini in a series of contributions to the International Journal of Microscopy and Natural Science, 1894–1895, entitled the 'Bacteria of the Sputa and the Cryptogamic Flora of the Mouth.' By a special method of staining he discovered the organism in the *materies alba* of Leuwenhoek.[1] Preparations were made before the first morning meal. This highly specialised form of *Leptothrix* is said to resemble the algæ. It is a long, thread-like micro-organism having segmentations or subdivisions, is uncoloured by iodine or weak acids, and at present is quite uncultivable. If the *materies alba* be examined, it will be found to contain, beside micrococci and bacilli, long, interlacing filaments, non-jointed, and non-coloured violet by iodine or acid. Vicentini wished to change the term *Leptothrix buccalis* to *Leptothrix racemosa* in order to represent its sporulation. This spore formation is only found in the superficial threads of the mass, the older filaments apparently being devoid of them.

"The female element consists of a central thread or stem, peduncles arranged in six rows, sporules and a gelatinous envelope. In other words *Leptothrix racemosa* is a thread-like organism found in the

[1] *Opera omnia sive arcana naturæ ope Microscopiorum exactissimorum detecta.* 1722.

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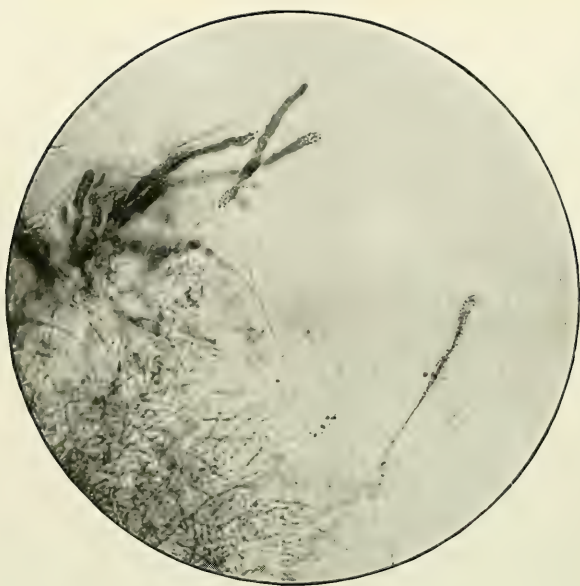
Fig. 383.—*Leptothrix racemosa.* (*Photomicrograph by Leon Williams.*)

Fig. 384.—*Leptothrix racemosa.* (*Photomicrograph by Leon Williams.*)

microbial placques of the interdental spaces during the hours of fasting, and has beaded stems which bear at their extremities fructifying heads containing six or more rows of spores (Fig. 385).

"Vicentini considered that the microorganism passes through four phases in its life history. First, that common to all bacteria, and undifferentiated from them; second, a stage of transition, in which

FIG. 385.—*Leptothrix racemosa.* "Fructification heads." From approximal surface of a tooth. (*Photomicrograph by Leon Williams.*)

there may be recognised 'chains, bundles, and masses of inter-twined filaments, isolated filaments, large dumb-bell bacteria, and masses of diplococci, the large dumb-bell bacteria being derived from the diplococci, the true original cocci linking together.' The third phase occurs with the deposition of tartar on the teeth. Here he described large threads, often massed together in bent, filamentous

forms, which presented the peculiar appearances of branched or barbed radial extremities and bulbous terminations. And in the last phase, which Vicentini called that of 'fructification' or 'complete aerial vegetation,' the fertile heads or extremities assume a more complicated appearance, and exhibit the formation of spores arranged in three longitudinal rows. 'The fertile filaments are sometimes straight, at others bent or curved, occasionally they are entirely wanting, because the fructifications have been carried away by mechanical force.' 'Gemmules of reserve' adhere to the periphery of the stem.

"Vicentini believes that in its method of reproduction this organism follows two methods—the endogenous spore formation, and the conjugated fructification or acrogenous abjunction, in which spores are found at the apices of the cryptogam. The terminal cell becomes enlarged and converted into a tiny base called the basidium, from which arise minute stalks or sterigmata, carrying spores upon their terminal ends.

"In this way it is possible that *Leptothrix racemosa* possesses veritable organs of reproduction and therefore resembles fungi and the diæcious algæ. The male elements have a morphological likeness to blossoms formed of spindle or fern-shaped points.

"Vicentini believed that this micro-organism is the parent of all but one of the varieties found in the sputum. Thus he asserts that *Bacillus buccalis maximus* and *Leptothrix buccalis* represent merely its broken stems and filaments, *Iodococcus vaginatus* the 'gemmules of reserve,' and the *Spirillum sputugenum*, the appendages or branches of the male elements.

"Much of this work is corroborated by Leon Williams, who, however, does not hold entirely with all his views."

THE MICRO-ORGANISMS OF DENTAL CARIES

The organisms found in dental caries are of various types; some active in the various stages of the process, others simply present as accidental invaders and of no significance as far as the condition is concerned.

Miller demonstrated conclusively that dental caries was caused essentially by bacterial action, isolating bacteria from carious teeth and furthermore producing all stages of caries artificially.

Goadby grouped the organisms of dental caries into three classes: namely those having the property of (A) forming acid, (B) of peptonising dentine; and (C) producing pigmentation. He also

mentions a number of organisms found in a group of cases studied. Our experience has been that the organisms encountered in the mouth and in the various stages of dental caries vary to a great extent and that by special methods of cultivation, some are noticed which ordinarily are not found. Therefore it seems out of place in this chapter to attempt to name all of the different organisms concerned in the process.

The organisms concerned in the various stages may be grouped as follows:

I. *Those entering into plaque formation.* Various types of cocci, bacilli, leptothrix and streptothriciæ. These have been considered in sufficient detail under the findings in stained preparations.

II. *Those concerned in acid formation.* To attempt to name and consider all organisms associated with dental caries would be inexcusable in this chapter; the following statement covers the entire situation. Any organism capable of developing in the oral cavity, and of forming acids by splitting carbohydrates, may play a part in dental caries by forming organic acids, the most important of which are acetic and lactic, which by their constant or intermittent action, notably on protected surfaces, underneath plaques, etc., disintegrate enamel. Butyric and succinic acids are also formed, and they are claimed by some to exert a similar action.

Some speak of *acid-producing organisms* in this connection and actually believe that the organisms produce acids. The facts are as follow: The micro-organisms produce enzymes of various kinds, some of which have the property of splitting up carbohydrates into simpler compounds, amongst which are the acids already named.

III. *Those disintegrating dentine:*

(A) Acid formers as above, the acid dissolving the lime salts in the dentine.

(B) Various organisms capable of digesting proteins. This might include any organism capable of developing in the oral cavity producing proteolytic enzymes or decarboxylases and deamidases. The above groups may be divided as follows:

(a) Those acting on the superficial layers of carious dentine.

(1) Aerobic: those acting (developing) only in the presence of free oxygen.

(2) Aerobic facultative: those acting both under aerobic and anaerobic conditions (Fig. 146).

(b) Those acting on the deeper layers of carious dentine.

(1) Aerobic facultative.

(2) Anaerobic: Those not capable of developing in the presence of free oxygen, but growing only under strict anaerobic conditions. The conditions in deeper caries, underneath masses of food-débris-filled cavities, and underneath temporary and permanent fillings, are ideal for their development.
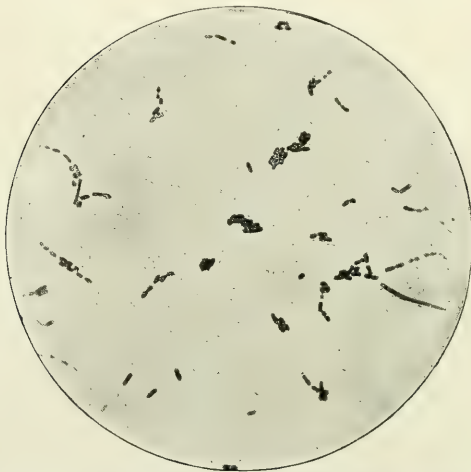
FIG. 386.—*Bacillus necrodentalis.* Showing pleomorphism. Forty-eight hours' cultivation of agar. Stained by Gram's method. (Magnified 1,000 times. (*Trans. Odonto. Soc. of Great Britain.*)

Bacterial proteolytic enzymes are of two types: (1) Of the nature of pepsin, carrying the cleavage of protein as far as the formation of peptone; (2) Of the nature of trypsin carrying the cleavage of protein so far as the formation of amino acids.

The so-called putrefactive changes in caries, made manifest by the presence of a decided aromatic odor, are due to the end-products formed by the deamination and decarboxylisation of the amino acids, chiefly some of the higher open chain acids and those of the benzene series. This action is exerted by various types of bacteria under anaerobic conditions.

The organisms in the deeper layers of carious dentine have been the subject of some investigation and much discussion. The pre-

vailing opinion is that they are not numerous. Goadby gives as quite constantly present an organism which he isolated and called the *Bacillus necrodentalis*, the *Streptococcus brevis*, and *Staphylococcus albus*. The first is an anaerobe, the other two facultative. Organisms of the so-called Moro-Tissier[1,2] group of intestinal bacteria have been found in the deeper layers of carious dentine by Kleigler,[3] Howe[4] and others. Their relationship to the process is apparently an important one.

[1] Jahresberichte für Kinderheilkunde, 1900 and 1905.
[2] *Annales de l'Institut Pasteur*, 1900.
[3] *Journal of Allied Dental Societies*, 1915.
[4] *Journal of Medical Research*, 1917.

PART III

THE EXTRA-ORAL DENTAL TISSUES

CHAPTER XVII

"DERMOID" TEETH, OR TEETH DEVELOPED IN
TERATOMATA

Relation of teeth to Teratomata—Varieties—Eruption—Development—
Shedding—Anatomy and Histo-pathology—Bony attachments—
Relation of "Dermoid" teeth to Hair—"Dermoid" teeth in the
testis—Conclusions.

"Dermoid" teeth, or teeth developed in teratomata, are of in-
terest to the embryologist, the pathologist, the surgeon, and, to
the dentist. Teeth appearing elsewhere than in the maxilla or the
mandible must be considered as abnormal. The origin of such
is now believed to be almost certainly connected in every case with
a teratoma. These teratomata occur most commonly in the ovary,
in the neck, and possibly in the testis. They are seen in the human
subject, and also in animals of a lower grade. When occurring in
the neck, a teratoma is probably an anterior dichotomy, abortive
in most cases. These remarks are confined to the dental structures
which are by no means infrequently discovered in those cysts not
uncommonly associated with the human ovary, and have been for
long termed "dermoid cysts."

In 1860, Salter[1] published an excellent account of these teeth,
while in 1863 Alfred Coleman[2] presented another. In 1890
T. Charters White and J. Bland-Sutton[3] jointly read a most in-
structive contribution to the subject, and later S. G. Shattock[4]
summarised in a brilliant article, the then-existing knowledge of the
subject.

" DERMAL " TISSUES

An ovarian "dermoid" is a true teratoma.

It is interesting to note that it is what may be called the "dermal"
tissues which seem to be most in evidence. There is abundance of
skin, often much hair, and this of two kinds, fine lanugo-

[1] *Guy's Hosp. Reports*, 1860.
[2] *Trans. Odont. Soc. of Great Britain* (1863–65), 1865.
[3] *Dental Record*, 1890.
[4] *Trans. Path. Soc.*, 1907.

like, and long and scalp-like, each growing from its own appropriate part of the fœtus, and teeth, sometimes only one, often many, and occasionally exceedingly numerous. Also in a few instances nails or horny structures have been observed.

RELATION OF TEETH TO TERATOMATA

The exact position of these teeth in relation to the teratomatous tissue and to the actual cyst wall is a matter of some interest and
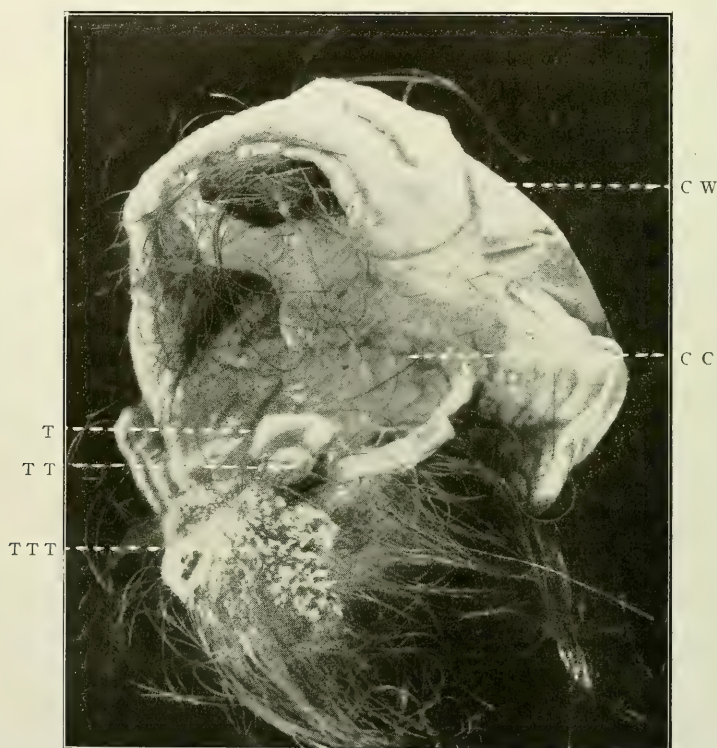
FIG. 387.—An ovarian teratomatous cyst laid open to show contents. c.w. Cyst wall; c.c. Cyst cavity; т. Caniniform tooth; т.т. Another portion of the teratoma, possibly an arm; т.т.т. Possibly the scalp.

importance. The teeth may be found (1) embedded in bone in actual alveoli or sockets, (2) embedded in soft tissue, either of the fœtus or possibly of the cyst wall, or (3) free within the cyst cavity.

There is no reason why a teratomatous fœtus should not possess an ill-formed maxilla or mandible, and no reason why such a jaw

should not carry teeth. Hence the appearance on, or in the tissue forming the fœtus, of bone with attached teeth is easy of explanation; but it is difficult to account for those instances where teeth have been found at some distance apparently from the actual site of the fœtal tissues, and attached to soft tissues only. There are three possible explanations: First, that during the development of the cyst, associated with which is the fœtus, pressure has caused a lateral displacement of a part of the fœtus, so that the tooth-bearing area has, as it were, become flattened out, leading to an elongation
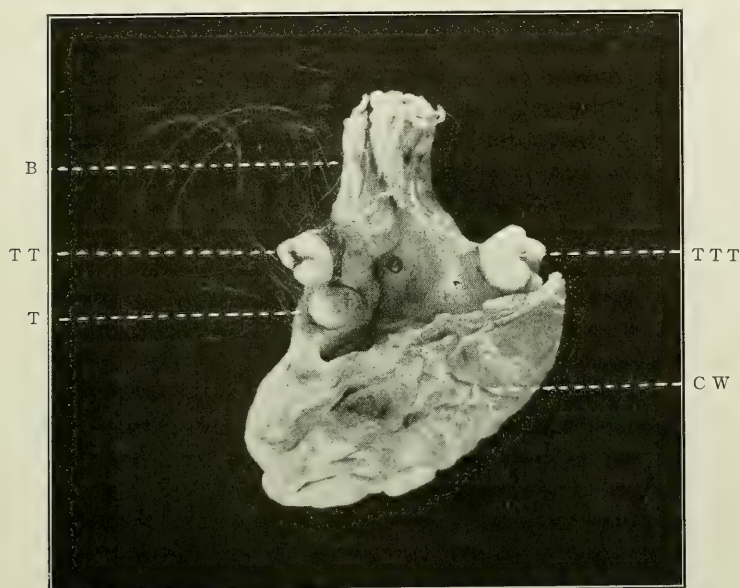
FIG. 388.—A portion of an ovarian teratomatous cyst laid open to show contents. c.w. Cyst wall; B. Bone to which teeth were attached; T. Caniniform tooth; T.T. Premolariform tooth; T.T.T. Molariform tooth.

of the tooth band. Secondly, there may have been a homologous twin which has developed very much less than its fellow, in fact, the teeth may be the sole evidence of its existence. It is possible for the same ovary to contain three separate teratomata, or triplets, and thus for the number of teeth present to be considerable, and their position remote in relation to the obvious teratoma. Or, thirdly, it may be an anterior dichotomy of the teratoma, thus allowing for at least a second dentition.

It is always the cephalic end of the teratoma in which these "der-

moid" teeth arise, and, in fact, the dental structures may in some instances be the only evidence of the cephalic end of a trunkless (acormous) teratoma.

VARIETIES

The morphological variations in teeth associated with ovarian teratomata approximate very closely to those usually found in the human mouth. Incisors, canines, premolars and molars have their counterparts in these cysts. The premolariform and caniniform types predominate from the point of view of number. The teeth themselves are generally well developed, and bear few, if any, traces of any degeneration of their exposed portions. Seldom, if ever, have purely conical representatives been noticed in these cysts. Sometimes, however, very numerous malformed dental bodies are met with. These possess but little resemblance to ordinary human teeth.

The cause of such enormous quantities of these denticles is difficult to determine. It may be that an extended tooth band has given rise to myriads of aborted, but more or less calcified, tooth germs. Or that fenestration and total disappearance of the intervening portions of the tooth band have occurred in the usual way, and that arrest of complete development, caused by the abnormal environment to which they have been subjected, coupled, perhaps, with the precocity and rapidity of their growth, has resulted in the display of congeries of misshapen, irregular masses composed mainly of enamel and dentine. Although these teratomatous cysts are tooth-bearing cysts, they are in no sense dentigerous cysts, and it would appear to be extremely likely that the same operations of pathogenesis may be acting here as in similar fashion to those which act sometimes in the jaws. It is not beyond the bounds of possibility that there may be occasionally a multiplicity of tooth-bearing cysts, which ultimately are capable of becoming incorporated in one large cavity.

DEVELOPMENT

In "dermoid" oöphoronic cysts "epithelial pearls" exist. The cells are large, become compressed as they approach the peripheral portion of the "pearl," and are finally lost in the surrounding capsule. They are ingrowths from the surface epithelium, which become isolated in the mesodermic tissue, and are allied to enamel organs, being attached to the free surface where they arose. Many

ovarian tooth germs lack a definite fibrous capsule.   (See Fig. 256, Vol. I.)

The so-called "pearls" may remain cellular, or give rise to the formation of enamel, or undergo transformation into horns or nails.

ERUPTION OR NON-ERUPTION OF TEETH

Of the actual dynamics of the eruption of ovarian teeth it is impossible to speak.   Many of the oral conditions which assist this phenomenon are entirely absent.   The growth of bone when present may have some bearing upon the eruption, but this cannot hold in those
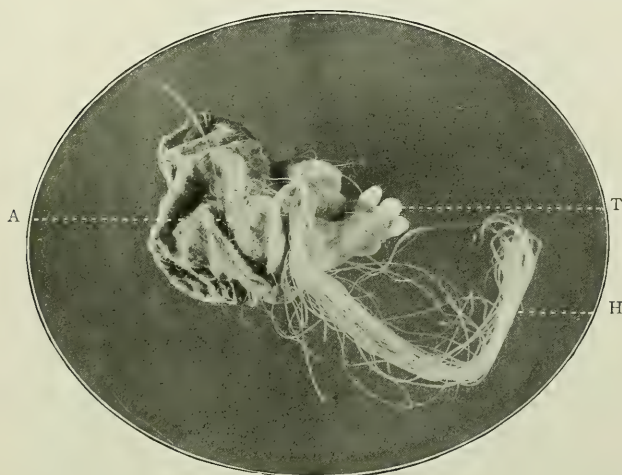
FIG. 389.—Part of the contents of an ovarian "dermoid" cyst.   T. Three well-formed premolariform teeth; H. A bundle of hair; A. A mass of teratomatous bone.   (*Photograph by Mr. Dencer Whittles.*)

instances in which teeth are erupted from the wall of the "dermoid" in places where no bone exists.

Many cysts contain enamel organs which have not proceeded to maturity.   It would seem that many teeth while fully developed, except perhaps as far as their roots are concerned, do not become extruded through the superficial soft parts, though evidences are not wanting that generally, by virtue of their rapid and precocious growth, they do completely erupt on the surface of the teratoma.

There is no evidence of any eruption of a second dentition such as occurs in the normal mouth.

SHEDDING

In the ovarian embryomata hair is frequently shed. The same cause may be at work in producing this separation as in the case of other epidermal derivatives. The implantation of the ovarian teeth is comparatively feeble in character. They are retained in the fresh condition by a thin annular elevation of connective tissue at their necks, and in many dried specimens there is an appearance as if the marginal bone had become absorbed, or, at all events, had never been fully developed. The necks of the teeth are well
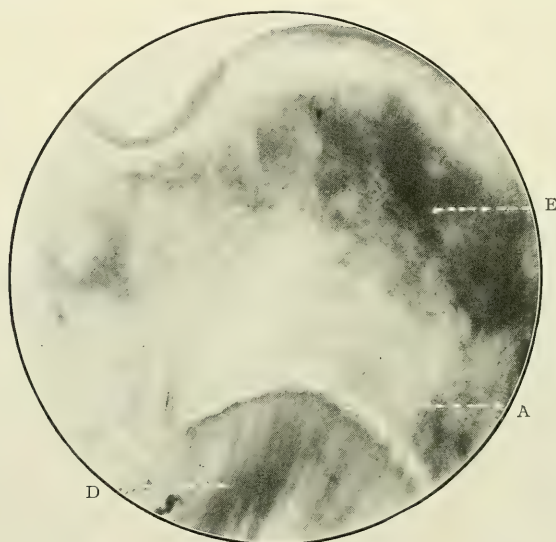
FIG. 390.—Vertical section of the enamel. Magnified 55 times. E. Enamel. D. Dentine. A. Amelo-dentinal junction.

exposed and vary in depth, and often their roots are visible above the free surface of the bony alveolus.

It is possible that when a tooth is found free within the cavity of the cyst, it has been shed by senile changes, in an acardiac, acormous parasite as part of a pathological retrogression which is entirely different from, and antecedent to, that of the host. But in other cases it may but be an accidental detachment during the removal of the cyst from the body.

ANATOMY AND HISTO-PATHOLOGY

For the most part the teeth found in ovarian teratomata exhibit the main characteristics of those of the human permanent denti-

tion, though on the whole they are smaller. In those examined they measured about 5.5 mm. in their extero-internal diameter (which would correspond to the bucco-lingual direction in the mouth) and 17 mm. in extreme length. But, of course, many variations of mensuration are met with. In those specimens specially examined for the purpose, the translucent pellicle of Nasmyth's membrane was found.
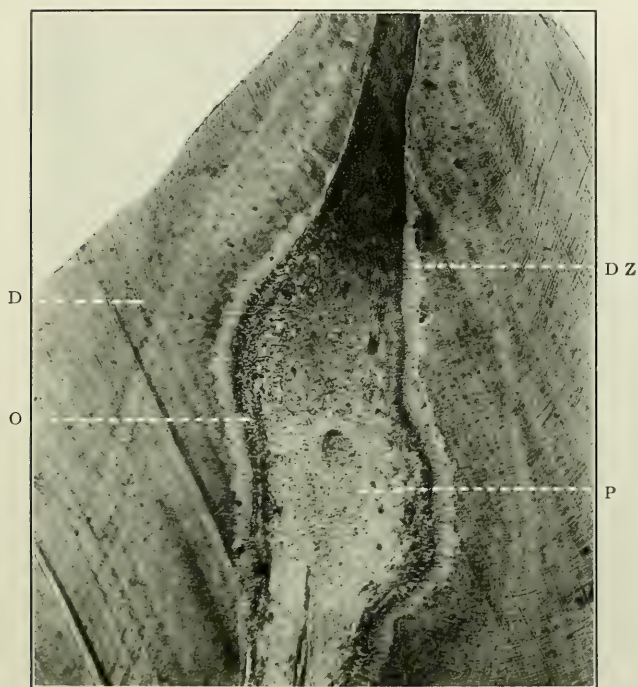
FIG. 391.—Vertical section of an ovarian tooth. Decalcified. Stained with hæmatoxylene. Magnified 45 times. D. Dentine; P. Pulp tissue; O. Odontoblasts; D.Z. Dentogenetic zone.

A root is generally present, being joined to the crown sometimes without the usual cervical constriction. Seldom is a tooth bi-rooted, and a multi-rooted tooth is very rare. The roots taper to a point.

On section a pulp cavity with root canal can usually be observed. In extremely thin incisiform specimens this is often narrowed down to an inconspicuous canal, and even this at times may be wanting. In the fresh condition pulp tissue is present, and bundles of myelinic nerve fibres can be seen accompanying the blood-vessels. When no

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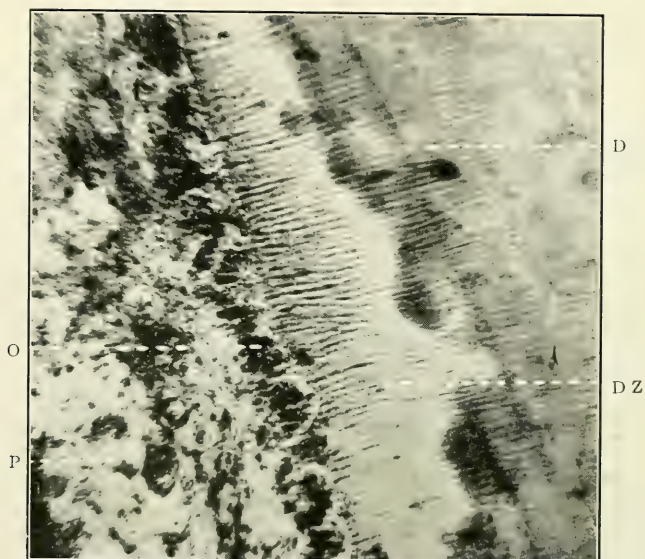
FIG. 392.—Section of an ovarian tooth with pulp *in situ*. Stained with hæmatoxylene. Magnified 200 times. D. Dentine. D.Z. Dentogenetic zone; P. Pulp; O. Odontoblasts.
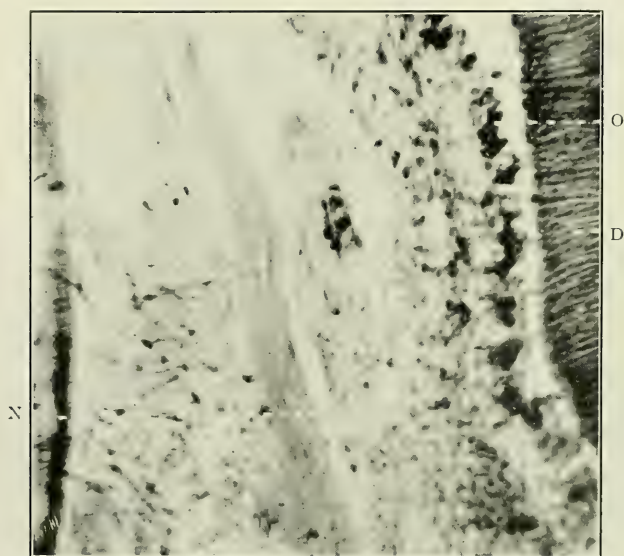
FIG. 393.—Similar to preceding figure. D. Dentine; O. Odontoblasts; N. Myelinic nerve fibres.

actual cavity exists, dentinal tubules radiate more or less from a common centre outwards, but in some sections, the tubules run centripetally as well as centrifugally (Fig. 65).

Enamel, dentine and cementum are present. The first is fairly normal, the second of an incompletely developed character, as proved by the abundance of interglobular spaces. Cementum is frequently absent. If it is present, it constitutes a very thin external band of the dentine, just beyond the homogeneous layer and the granular layer of Tomes. The pathology of these teeth has been cursorily described by Wedl (Atlas der Path. der Zähne, 1903). He believes that erosion of the superficial parts may take place. He
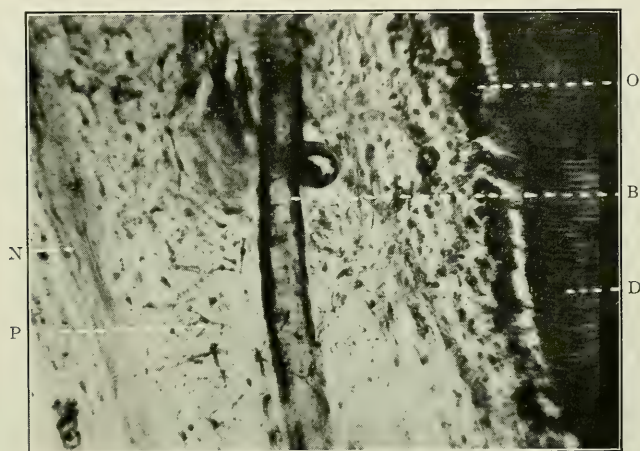
FIG. 394.—Similar to preceding. D. Dentine; P. Pulp; O. Odontoblasts; N. Myelinic nerve fibres.

has not seen dental caries, which is to be expected, inasmuch as the contents of the embryomatous cyst are generally of a thick alkaline nature, and suppuration often occurs. Mr. Shattock says that enamel nodules may sometimes be seen upon the roots. The teeth are clumped together, and are malplaced when found embedded in the teratomatous bone. Instead of occupying a definite relationship to each other, as in the mouth, they are placed irregularly with regard to one another, possibly from the fact that here there are no mechanical factors such as are produced by the action of the soft tissues of the tongue and cheek or lips in giving rise to the proper alignment of the teeth in the dental arch.

BONY ATTACHMENTS

Some of the pieces of bone in which these ovarian teeth have been found are exceedingly like small maxillæ or ill-developed mandibles. One peculiarity, however, is often present, namely, that whereas the size of the "jaw" itself is much less than the size of the jaw of the host, the teeth themselves found in the ovary may be almost, if not quite, as large as those found in the normal mandible of the host. The bone itself is of poor quality, and consists mainly of cancellous tissue.

RELATION OF "DERMOID" TEETH TO HAIR

As a rule the teeth in the teratomata of the ovary bear no relationship to the hair growing from the same parasite, other than that they will be found in their own normal position in reference to the cephalic end of the teratoma, and the long hairs will be springing from the scalp portion of the fœtus, also at the cephalic end. But sometimes short, rather stubbly hairs are found growing, in a ringlet, actually round the neck of the tooth from the soft tissue which might be said to be forming the gum. This fact is interesting as showing the close possible connection between the two dermal structures, hairs and teeth.

X-RAY EXAMINATION OF "DERMOID" TEETH

The X-ray examination of "dermoid" teeth is interesting from several points of view. The teeth, like normal teeth, obstruct the passage of the rays more markedly than does the bone by which their roots may be surrounded. Hence it is quite possible that an X-ray examination of a living subject who is the host of a dermoid cyst of the ovary might reveal the presence of the cyst by the marked shadow thrown by the teeth, if any were present, in contrast to the shadow thrown by the pelvic bones.

A radiograph will also indicate the presence of a pulp cavity in the interior of the "dermoid" teeth. It will also show the character of the bone associated with the teeth, if any bone exists.

"DERMOID" TEETH IN THE TESTIS

There are on record several cases of a dermoid cyst of the testis in the human subject, but the condition must be considered as an

extremely rare one. A similar cystic enlargement of the testis of the horse is not so uncommon, and particularly where the testis is retained. One case at least, occurring in the human subject, presented a tooth borne by a teratoma in the testis. In the case of the horse there have been found similar teeth.

While the origin of ovarian "dermoids" may now be considered as settled, it is difficult to conceive that the testis can be the host of a teratoma with an identical origin. Prof. Shattock has put forward the suggestion that these "dermoids" of the testis are in reality teratomata of the ovary portion of an ovi-testis gland in a true hermaphrodite. This is certainly quite feasible, and some amount of confirmation is obtained from the presence of a teratoma associated with an imperfectly descended testis in a "rig" horse. Such an animal is not infrequently vicious and unsuitable for domestic purposes. The removal of the retained testis has the effect in many instances of rendering the animal docile and useful.

CONCLUSIONS

The presence of teeth in dermoid cysts of the ovary, and of the testis, tends to prove the teratomatous nature of these tumours.

The similarity between dermoid cyst teeth and those of the normal human mouth tends further to indicate their teratomatous origin.

The period of eruption of the "dermoid" teeth does not coincide with the period of eruption of the teeth of the host, but is probably earlier, and their growth is more rapid. It is possible that this precocity may be due to the influence of the super-host through her blood.

There is no distinct evidence of the shedding of "dermoid" teeth, and there is no evidence of any distinction between a deciduous and a permanent dentition.

While "dermoid" teeth may be ill-shapen and may otherwise deviate from the form of normal teeth, there is no evidence of any pathological process which can be termed "caries."

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